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CLINICAL AND EXPERIMENTAL RESEARCHES ON INTRAOCULAR DRAINAGE.[1]

A PRELIMINARY REPORT.

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I.—INTRODUCTION

EXPERIMENTAL researches and clinical studies on intraocular pressure may be made from entirely new points of view in consequence of the introduction into ophthalmic practice of the Schiötz tonometer.

The advantage of the measurement of intraocular pressure by means of the Schiötz tonometer consists in its applicability to clinical work. One is thus enabled to get data more accurate and reliable than have been obtained in the laboratory, where the measurement is made on animals by connecting with a manometer a canula or hypodermic needle introduced into the anterior or posterior chamber of the eye.

The introduction of a canula into the eye means a severe trauma to this most delicate organ. Such a trauma, I believe, must disturb at least for a certain length of time, the entire mechanism of circulation of intraocular fluids. Even the mere application of the tonometer on the eye, doubtless, produces a certain amount of traumatism, but this is inconsequent compared with that due to entering the eyeball with a canula or a needle. During the past year and a half, I have utilized the tonometer of Professor Schiötz for the examination of intraocular pressure in the eyes of human subjects and of

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rabbits and cats, and I am convinced that the results are more reliable and exact than would have been obtained if I had used the tonometer of Schiötz in human eyes and the canula and manometer in the eyes of animals.

Since Prof. Schiötz's second article on tonometry (*Arch. f. Augenheilk.*, vol. lxii., 1909; these ARCHIVES, vol. xl., 1911) a number of very valuable contributions discussing various questions in connection with the intraocular pressure has appeared. Some of the questions, as stated above, have been partly cleared up, but many have been left untouched. One of these is the following: **What is the normal rate of drainage of intraocular fluids in animal and human eyes, and how is this rate of drainage affected in glaucomatous eyes?**

All who have had experience with the tonometer have observed that when this instrument is continuously applied on the eye for a certain length of time, or at very short intervals, it registers a gradually decreasing o. p. (intraocular pressure)—*i. e.*, the eye becomes gradually softer. The rational explanation of this phenomenon is that the weight of the tonometer expresses a certain amount of fluid from the eye, thereby reducing its hardness. It is interesting to note that this fact has not been utilized in studying the drainage system of the normal and glaucomatous eyes. If by applying a weight on the eyeball, it is possible to express from this organ a certain amount of fluid, it is evident that there are channels in the eye through which this fluid is expressed.

It is almost universally admitted that the intraocular fluids are constantly renovated by a continuous slow flow of liquid which enters the eyeball and also by a continuous, just as slow exit of liquid. Weiss (*Zeitschr. f. Augenheilk.*, vol. xxv., 1911) stands almost alone among ophthalmologists in asserting his doubt whether normally there is a constant, continuous circulation going on in the fluid contents of the eyeball. From the time of the researches of Leber and his pupils down to the latest work on this subject by Wessely (*Zeitschr. f. Augenheilk.*, vol. xxv., 1911) it is practically admitted by all ophthalmologists that normally fluid drains from the eyeball at the same rate at which it flows into the eye, that the "intraocular pressure is obviously a function of the volume of the contents of the globe and that the variations in internal

pressure must be due to changes in the amount of fluid (blood and lymph)."—(Parsons's ***The Pathology of the Eye.***)

Wessely (*loco cit.*) even figures out that the aqueous humor is drained and completely renovated every two hours. It is not within the range of this paper to describe the paths of drainage of the ocular fluids. It does not concern us, at this moment whether the draining function of the eye is taken care of by Schlemm's canal, the iris, the ciliary processes, or by the choroidal vessels, or by all together. The main question is that normally there is taking place in the eyeball a constant drainage of its fluid contents. As the intraocular pressure is maintained at a certain level by the constant volume of the contents of the globe, it is obvious that this constant intraocular pressure is possible only as long as the amount of liquid entering the eye does not exceed the amount leaving it. In eyes in which the draining function is more or less interfered with, there is a loss of balance between the two main factors (in and outflow) regulating the intraocular pressure. The ocular fluid cannot leave the eye at the same rate as it does normally and the fluid contents of the eyeball will have a tendency to increase in amount, consequently the intraocular pressure will increase also. Supposing that a weight would be constantly applied on an eye with its drainage system in good order, that weight will gradually express from that eye a certain amount of fluid in a certain number of seconds. If the same weight is applied on an eye with its draining system partially obstructed (simple glaucoma), it will take a longer time to express the same amount of fluid. Finally, if the same weight is applied on an eye with its drainage entirely obstructed (absolute glaucoma), no matter how long we wait no fluid is expressed from that eye. Imagining that instead of the weight we use the tonometer, we readily see how we can judge at the same time the amount of fluid expressed from the eye by reading off from the tonometric scale the degree of decrease of intraocular pressure when the tonometer is applied steadily for a certain time. In a normal eye the tonometer shows that the intraocular pressure is gradually decreasing at an average which we call normal; in an eye with an impaired drainage the weight of the tonometer still expresses fluid but at a much slower rate, and in an eye with absolute

glaucoma the weight of the tonometer produces no decrease of the intraocular pressure.

From the above, it is seen that the tonometer may be utilized for the accurate measurement, not only of the intraocular pressure, but also of the index of ocular drainage.

The index of ocular drainage may be defined, in general terms, **as the rate or rapidity with which the ocular fluid may be expressed by the weight of the tonometer applied on the eye.**

Since I have begun to measure the ocular drainage in animal and human eyes, I have become convinced that the mere measurement of intraocular pressure, while very helpful in the diagnosis of certain cases of glaucoma, is a procedure which may be incomplete and misleading unless the index of ocular drainage is measured at the same time. It is misleading in those cases which have only occasionally a high intraocular pressure. If the tonometer happens to be applied during one of the intervals when the o. p. is not above the normal limit, we may be induced to believe that the eye is normal. In fact, clinicians no longer rely upon a single examination. In suspicious cases, they have the o. p. measured repeatedly. It is not improbable that in some cases the examinations should be made during the intervals when the intraocular pressure is not high. In such cases, the mere taking of the o. p. with the tonometer is a crude and incomplete procedure.

K. Heilbrun (*Arch. f. Ophthalm.*, vol. xxix., 1911) and Stock (*Beilageheft zu Klin. Monatsbl. f. Augenheilk.*, 1910) have each published histories of patients who had low intraocular pressure during attacks of glaucoma. My experience fully agrees with that of Heilbrun who says: "The tension, which lies within the normal limits, may be abnormally high in cases which have had an abnormally low intraocular pressure before." The mere satisfaction of proving that the intraocular pressure of an eye is below 27*mm* Hg., *i.e.*, within normal limits, is sometimes deceiving and such an examination is incomplete. On the one hand, a low intraocular pressure does not exclude the presence of glaucoma, because the examination may happen to be made during an interval of non-irritability or because the original o. p. of the eye, when normal,

was very much lower than the one now found. On the other hand, the measurement of the o. p. is interesting only in so far as it reveals the condition of the safety devices which such an eye possesses for keeping its draining system in good order. In other words, the measurement of the intraocular pressure alone throws some light upon the condition of the drainage system of the eye. This light is very dim, however, because the drainage capacity may be impaired to some degree and still the o. p. may not exceed the normal limit. (We report such instances in the second part of this paper.) Therefore, a correct idea about the normal condition or about the state of impairment of the mechanism regulating the intraocular pressure, can be obtained only when we consider both the intraocular pressure and the index of drainage of the ocular fluids. The elucidation of this point is of great importance in the early diagnosis of glaucoma, when the various complaints of the patient are vague and indefinite and the information furnished by a most careful examination is very doubtful.

I believe that the diagnosis in doubtful cases of incipient glaucoma might be cleared up at a stage when the ophthalmoscope, perimeter, and Bjerrum's curtain, etc., are of no avail.

The question of intraocular drainage measured on the eye in perfect physiological integrity (not on eyes punctured by a canula) has not been studied. The normal rate of drainage in human and animal eyes, the index of drainage in glaucomatous eyes at various stages, the influence of myotics, mydriatics, and cycloplegics on the drainage of ocular fluids, and the effects on the drainage of various surgical operations on the eye are some of the problems concerning the question of intraocular pressure.

We have examined the intraocular pressure and rate of drainage in 3 eyes of rabbits, in 7 eyes with normal o. p., in 3 eyes with suspicious glaucoma, in 4 eyes with simple glaucoma, in 4 eyes in which an iridectomy had been performed, in 2 eyes with simple glaucoma and retinal hemorrhages, in 2 cases of absolute glaucoma, in 1 with absolute glaucoma in which Dr. O. Schirmer had performed a resectio optico-ciliaris, and in 1 with secondary glaucoma and chronic irido-cyclitis.

As to the method of examination for the index of ocular

drainage, a correct technic is necessary and all possible precautions must be taken in order to avoid errors. The method which I have used in examining the rate of ocular drainage is as follows:

For rabbits, an assistant takes the rabbit on his lap and holding it stretched, fixes the head in such a manner that one eye is directed as nearly straight upward as possible. Another assistant, with a watch before him, records the initial intra-ocular pressure shown by the tonometer and the exact time in seconds at which the examination is begun. He then notes the number of seconds it takes the handle to move from the one division of the tonometric scale to the next one. The examination ends when the animal becomes restless or when the handle of the tonometer does not move while the instrument is applied for over 120 seconds. In human eyes the examination is made with the patient lying on a couch or a table.

II

INTRAOCULAR DRAINAGE IN RABBITS.[1]

RABBIT I. Intraocular Pressure and Ocular Drainage Measured in the Right Eye. Weight Used 7.5 gr.

December 20, 1911		December 20th Experiment repeated after 30 minutes		December 25th[2]	
	mm Hg		*mm* Hg		*mm* Hg
O. P.	22.	O. P.	26.		
30″ later	18.5	15″ later	22.		
120″ "	16.	100″ "	18.5		
30″ "	13.	60″ "	16.	O. P.	16.
30″ "	16.	90″ "	13.	25″ later	13.
25″ "	13.				
20″ "	11.5	10″ "	11.5	65″ "	11.5
20″ "	13–16.				
20″ "	13.				
		15″ "	10.	20″ "	10.
		170″ "	8.5	60″ "	11.5
		90″ "	6.5	95″ "	13.
				27″ "	10.
				53″ "	6.5 to 8.5
				15″ "	6.5
				25″ "	6.5

(*Note*: ″ means seconds.)

The ocular drainage measured while the rabbit was under general ether anæsthesia gave following result:

O. P. 18.5[3]
5″ later 16.
300″ " no change

Rabbit I. Intraocular pressure and ocular drainage measured in the left eye. Weight used 7.5 (unless otherwise stated).

Nov. 14, 1911. O. p. (under ether general anæsthesia) 26 to 30.5.

Jan. 3d. Ocular drainage test under local (holocain) anæsthesia, weight 5.5.

[1] Numerous measurements have been made; for the sake of brevity we reproduce only a few which illustrate the rate of ocular drainage.

[2] On the 21st of December I aspirated 0.5*cc* of vitreous from the eye and injected an equal quantity of physiologic salt solution. This explains why we find on Dec. 25th the intraocular pressure somewhat reduced.

[3] These numbers throughout the paper refer to *mm* Hg.

O. P.	6
8″ later	5.5
75″ "	4.5

Jan. 4th. Ocular drainage test about the same result.

Vitreous was aspirated for a purpose not connected with the subject of this paper and also in order to ascertain the changes of the intraocular pressure when vitreous is aspirated and replaced by physiological salt solution.

REMARKS: (A) The intraocular pressure decreased about 13*mm* Hg. after 0.4*cc* of vitreous had been aspirated and physiological salt solution injected instead.

(B) Under the influence of the (ether) general anæsthesia, the intraocular pressure increased from 6.5 to 22*mm* Hg.

(C) The measurement of the intraocular drainage on this eye revealed the interesting fact that in spite of its initial very low intraocular pressure (Jan. 3d and 4th) it took one day, 83 seconds to reduce the o. p. from 5.5. to 4.5, and the next, 176 seconds to reduce the o. p. 1.5 from 6.5 to 5.

Rabbit 2. Drainage of fluids of the right eye examined. Holocain used as a local anæsthetic. Weight used 7.5.

December 22d		December 22d (30 min. later)	
O. P.	30.5	O. P.	30.5
25″ later	26.	135″ later	26.
35″ "	22.	105″ "	22.
100″ "	18.5		
90″ "	16.		

December 22d (30 min. later)		January 3d.	
O. P.	18.5	O. P.	22.
55″ later	16.	55″ later	18.5
15″ "	13.	12″ "	16.
		33″ "	13.

Remarks: We see the same apparent irregularity of the rate of ocular drainage as in the right eye of Rabbit 1. It is also important to note that during the time I have been studying the ocular drainage in the right eye of Rabbit 2, the left eye was passing through a panophthalmitis, due to an infection after an attempt to aspirate some vitreous. The evolution of the panophthalmitis did not seem to influence the o. p. or the index of the ocular drainage in the other eye.

I am not prepared to say why these very wide oscillations

occur in the index of drainage of the intraocular fluids, not only in different eyes, but even in the same eye. It seems logical to expect that the same weight applied on the same eye will press out the same amount of fluid each time, in the same length of time. In reality it is not so, and the above tables, though very few, are suggestive with regard to the variability and relative instability of the draining mechanism of the eye.

From this apparent inconstancy of the index of drainage of intraocular fluid, one important observation may be made, namely: **in all the eyes examined, drainage was present and fluid was expressed each time the tonometer was applied for a certain length of time.**

It will be seen later that this is not so in glaucomatous eyes.

III.—MEASUREMENTS OF OCULAR DRAINAGE IN HUMAN EYES WITH NORMAL INTRAOCULAR PRESSURE.

On account of the readiness with which patients with optic atrophy, submit themselves to repeated examinations, I have measured the intraocular pressure and the ocular drainage in three patients with optic atrophy. Their intraocular pressure does not seem to differ from that of perfectly normal eyes and the findings have given me valuable data with which to compare the tables furnished by numerous examinations of the intraocular pressure and of the index of ocular drainage in six glaucomatous patients.

PATIENT 1.—K. A., 65 years of age. Complete optic atrophy in the right eye and incipient optic atrophy in the left eye. V. O. D. = perception of light. V. O. S. $= \frac{20}{50}$.

The intraocular pressure taken at various intervals during the time between May, 1911, and January, 1912, was found to vary between 16 and 26*mm* Hg.

The ocular drainage measured gave following results:

Jan. 8, 1912		Jan. 15, 1912		Jan. 15th (5 min. later)	
O. D.	16.	O. D.	16.	O. D.	13.
49″ later	13.	15″ later	13.	20″ later	11.5
62″ "	11.5	65″ "	11.5	70″ "	10.
52″ "	10.			45″ "	8.5

About a similar rate of drainage was found by repeated examinations on January 19th, April 1st, and June 19th.

During the time the patient was under observation, the

vision decreased gradually to zero in the right eye and to ability to distinguish movement of fingers in the left eye.

PATIENT 2.—Fr. V., 64 years old, tailor. Tabes, double optic atrophy (incipient). V. O. D. $=\frac{20}{40}$; V. O. S. $=\frac{20}{40}$; o. p. in both eyes varied during the first five months (Dec., 1911, to April, 1912) between 16 and 26.

OCULAR DRAINAGE TEST

Jan. 22, 1912		2 minutes later	
O. P. O. D.	18.5	O. P. O. S.	16.
15″ later	16.		
80″ "	13.	55″ later	13.

PATIENT 3.—Ewald W. baker, 42 years old. Retrobulbar neuritis with incipient optic atrophy of both eyes. V. O. D. $=\frac{20}{200}$; V. O. S. $=\frac{8}{200}$.

The intraocular pressure in each eye was between 16 and 26 during the first twelve months he was under observation.

OCULAR DRAINAGE TEST

O. P. O.D.	18.5	O. P. O. S.	11.5
30″ later	16.	104″ later	10.
23″ "	13.5		
53″ "	11.5		
90″ "	10.		
47″ "	10.		

From the tables of ocular drainage tests made on these patients with optic atrophy (and with normal intraocular pressure) we see:

(1) That in these eyes, as in rabbit's eyes, the time required for a given amount of intraocular pressure to descend a certain number of millimeters of mercury is not the same in every patient.

(2) The same eye of the same patient does not exhibit the same index of drainage when examined at various intervals. (See tables.) In the first patient, the intraocular pressure went down from 13 to 11.5 in 62 seconds on January 8th, in 65 seconds one week later, in 70 seconds five minutes later, etc.

IV.—MEASUREMENT OF INTRAOCULAR PRESSURE AND DRAINAGE IN GLAUCOMATOUS EYES.

GROUP A—SIMPLE GLAUCOMA.

PATIENT 4.—A. K., 60 years of age. Chronic simple glaucoma. Vision with myopia corrected, O. D. $=\frac{10}{18}$; O. S. $=\frac{5}{10}$.

On Jan. 31, 1912, the intraocular pressure was O. D. 82, O. S. 85. After the use of eserine for two days the o. p. fell to O. D. 22.5, O. S. 18.5.

During twelve months the intraocular pressure varied between 10 and 16 when the patient used a myotic. As soon as he stopped the use of pilocarpine in either eye the o. p. rose rapidly to 50 and 60.

Measurement of the ocular drainage.

Nov. 2, 1912. Patient stopped the use of pilocarpine in O. S. for last two days.

O. P. O. D.	11.5	O. P. O. S.	70.
20″ later	10.	120″ later	no change
70″ "	8.5		
70″ "	6.5 to 8.5		

Nov. 16, 1912. Patient had been using pilocarpine 1% solution, one drop *t. i. d.* in each eye.

O. P. O. D.	11.5	O. P. O. S.	18.5
30″ later	10.	8″ later	16.
		15″ "	13.
		15″ "	11.

Nov. 8, 1912. Has been using pilocarpine 1% in both eyes; stopped using the drops in O. S. for two days.

O. D.	26.	O. S.	50.
48″ later	22.	135″ later	no change
120″ "	18.5		

I instilled one drop of 1% solution of pilocarpine once in O. D. and every ten minutes for one hour in O. S. I then measured the ocular drainage.

O. P. O. D.	13.5	O. P. O. S.	13.5
35″ later	11.5	25″ later	11.5
20″ "	10.	37″ "	10.
50″ "	8.5	57″ "	8.5
50″ "	6.5	53″ "	6.5
55″ "	6.	55″ "	6.
25″ "	no change	30″ "	no change

Remarks: The drainage system of the eyes of this patient seems to be in perfect order as long as a myotic is used and the pathological changes in the eye, wherever they may be, are very readily influenced by the use of pilocarpine. Parallel with the decrease of intraocular pressure we find an increase in the rapidity of ocular drainage. The tables under date Nov. 2, 1912, and Nov. 8, 1912, demonstrate very plainly how the ocular drainage suffers and grows less and less when the use of the myotic is stopped.

The readiness with which the ocular drainage is normalized and the intraocular pressure is reduced by the use of pilocarpine is strikingly indicative of the way these particular eyes will stand glaucoma. In this patient, neither the field, nor the acuity of vision has suffered during the year we have kept him under observation.

GROUP B. CASES WITH ABSOLUTE GLAUCOMA IN WHICH OCULAR DRAINAGE HAS BEEN MEASURED.

PATIENT 5.—D. R., 65 years old. O. D.: Absolute glaucoma. Resectio optico-ciliaris. O. S.: Chronic glaucoma, incipient cataract. Patient has been using 1% solution of pilocarpin in the left eye for several months.

Mar. 12, 1912. Ocular drainage test:

O. D.	36.	O. S.	22.
60″ later	no change	30″ later	16–18.5

We made the drainage test of the right eye at various times and obtained the same result: the pressure did not change no matter how long the tonometer was held on the eye. We hope to have occasion in the future to examine the drainage capacity of several glaucomatous eyes with resectio optico-ciliaris. This may throw some light on the question whether the intraocular fluids have some means of drainage via the optic nerve and its vessels.

PATIENT 6.—H. L., 58 years of age. Arteriosclerosis; glaucoma simplex, O. D. Multiple retinal hemorrhages.

Mar. 27, 1912. Patient has been using for several days 2% solution of pilocarpin drops in both eyes.

Ocular drainage test:

O. D.	36.5	no change after 60 seconds.
O. S.	42.	" " " " "

April 1, 1912. Patient has stopped pilocarpin drops for twenty-four hours. Ocular drainage test:

O. D.	30.5	155″ later	26.
O. S.	36.	175″ "	30.5

Instillation of pilocarpine 2% drops in each eye every five minutes for 45 minutes then ocular drainage tested:

O. D.	36.5	120″ later	30.5
O. S.	50.5	17″ "	42.
		75″ "	no change

Remarks: On March 27th, the drainage was nil. The application of the tonometer (weight 10) for 60 seconds did not reduce the tension although the patient had used 2% pilocarpin for several days previously.

On April 1st the ocular drainage was still very deficient since it took the o. p. of the right eye 155 seconds to recede from 30.5 to 26, and that of the left eye 175 seconds to recede from 36 to 30.5.

PATIENT 7. Morris R., 59 years old. O. D.: Old irido-cyclitis, secondary glaucoma. O. S.: Chronic simple glaucoma. Coloboma iridis artefactum.

V. O. D. =0. V. O. S. $=\frac{10}{200}$.

April 21, 1912. O. p. O. D. 62. O. p. O. S. 42.5 to 50.5.

April 23, 1912. Patient has used pilocarpine 2% solution drops in both eyes three times daily, O. p. O. D. 42.5. O. p. O. S. 36.5–42.

Ocular drainage test: After having the tonometer applied on each eye for 60 seconds, the o. p. remained unchanged.

April 25th. The patient has been using pilocarpin 2%, one drop every one or two hours. O. p. O. S. 36.5 to 42.5. Drainage test, no change after 60 seconds. In this patient as in Case 6 of this group the ocular drainage could not be influenced by the use of myotics.

PATIENT 8. A. N., 58 years old. O. D.: Chronic glaucoma; coloboma iridis artefactum. O. S.: Absolute glaucoma.

March 3, 1911. O. p. O. D. 62.5*mm* Hg. O.p. O. S. 85.5. The frequent use of pilocarpin, 2%, until April 11, 1911, reduced the o.p. to O. D. 36.5, O. S. 50.5.

Drainage test: No change of tension in either eye during 100 seconds, while the tonometer was continuously applied.

Remarks: In this case, contrary to what we have seen in patient 1, the use of myotics could not re-establish a normal ocular drainage.

GROUP C. MEASUREMENT OF OCULAR DRAINAGE IN PATIENTS WITH SUSPICIOUS GLAUCOMA.

PATIENT 9. Meyer R., 68 years old. O. S.: Absolute glaucoma; coloboma iridis artefactum. O. D.: Slight

hyperopic astigmatism. Glaucoma (?). Intraocular pressure at first was:

O. D. 18.5 to 22
O. S. 36.5 to 39.5

Drainage test, Feb. 7, 1912:

O. P. O. D.	18.5	O. P. O. S.	30.5
25″ later	16.	33″ later	26.
45″ "	13.	90″ "	no change
40″ "	11.5		

Mar. 22, 1912. Patient has been using pilocarpine drops in the left eye since February 7, 1912. He is complaining that once in a while the right eye feels uneasy.

O. P. O. D.	18.5	O. P. O. S.	30.5
60″ later	no change	50″ later	26.

Sept. 4, 1912:

O. P. O. D.	22.	O. P. O. S.	23.
120″ later	18.5	60″ later	20.
		60″ "	no change

It is interesting to note that the right eye with the same intraocular pressure (18.5) on February 7th and March 22d, exhibited a normal capacity of drainage at the first examination and almost a complete stoppage during the second examination. The complaint of the patient of feeling an uneasiness in the right eye (the good eye) and the momentary impairment of the normal drainage are the only points leading us to suspect a prodromal stage of glaucoma in the good eye. Otherwise, neither the ophthalmoscope and field of vision nor the measurement of intraocular pressure would give us any help in the diagnosis.

PATIENT 10. L. B., 65 years old. General arteriosclerosis neurasthenia. O. D.: Absolute glaucoma. O. S.: Glaucoma(?).

Aug. 16, 1911. O. P. O. D. 62. O. P. O. S. 16.

Mar. 2, 1912. Patient complains of occasional slight pains and blurring of vision in the good eye. The measurement of the ocular drainage shows:

O. P. O. D.	60.	O. P. O. S.	26.
50″ later	no change	10″ later	22.
		65″ "	22.

Apr. 10. 1912. Patient complains that her sight in the good eye (left) becomes dim occasionally and the eye hurts her sometimes. Has been using pilocarpin 1% drops on each eye until two days ago when the myotic was discontinued.

V. O. S. counts fingers at 30 feet (maybe more; patient is illiterate and stupid). Field of vision normal.

Measurement of ocular drainage:

O. P. O. S.	18.5
60″ later	no change

Remarks: In this case the question was whether the left eye is just starting on the same glaucomatous career as the other eye (with absolute glaucoma) did two or three years ago. Aside from the very indefinite complaint of the patient, that her vision is occasionally dim, we had no reason to think beginning of a glaucoma in the good eye. The acuity and field of vision, as far as could be determined in this patient, were normal and not receding during the ten months we have had her under observation. The pupil reacted well to light and accommodation and the fundus showed nothing abnormal. The i. o. pressure was always "within the normal limits" (16–26). The only disquieting phenomenon was the impairment of the ocular drainage on March 2 and April 10, 1912.[1]

PATIENT 11. J. H., 36 years old, nurse, on Feb. 9, 1912, complained that she has suffered from headaches for about three months and that the pains are most severe around the left side of the forehead and temples. Sometimes she sees various colors around the gas flame, if she closes the right eye. V. O. D. $=\frac{20}{30}$ plus; O. S. $=\frac{20}{30}$. Field of vision for white about 20 degrees concentrically contracted in the right eye and about 30 degrees contracted in the left eye. On the nasal side (left eye) field contracted to about 10 degrees from the fixation point. The error of refraction was carefully corrected. Intraocular pressure felt with the finger seemed normal. With tonometer O. D. 22, O. S. 26–30.5. Both disks showed a deep physiologic excavation.

Feb. 26, 1912. Patient has been using one per cent. solution of pilocarpine in the left eye until the night before, when advised by a neighbor she went to another physician to have her eyes examined. A mydriatic was used and the patient came next day to the clinic with both pupils widely dilated.

[1] I saw the patient again during Jan., 1913, suffering from an acute glaucoma of the left eye.

Ocular drainage:

O. P. O. D.	22.	O. P. O. S.	26.
15″ later	16.	60″ later	22.
60″ "	no change	60″ "	no change

The drainage capacity of these eyes does not seem to be normal. In rabbit's eyes and in normal human eyes, the O. P. shows a greater readiness to descend while the tonometer is being steadily applied, than was noted in this patient's eyes. In these two patients the intraocular pressure as given by the tonometer was within the normal limits. Yet, the rate of ocular drainage, another element just as important, if not more so, was not normal.

March 8th. Patient has used 1% solution of pilocarpin drops in the left eye.

Ocular drainage:

O. P. O. D.	16.	O. S.	22.
35″ later	13.	65″ later	no change
25″ "	11.5		

Drainage has improved in the right eye, but not in the left one.

March 22d For four days patient has discontinued the pilocarpin drops in the left eye.

Ocular drainage:

O. D.	18.5	O. S.	22.
45″ later	16.	45″ later	18.5
30″ "	13.5	45″ "	no change

The left eye shows right along less capacity for drainage and a higher intraocular pressure than the right eye.

Mar. 29, 1912. Patient has not used pilocarpine drops for about 11 days.

Ocular drainage test:

O. P. O.D.	13.	O. P. O. S.	22.
40″ later	11.5	75″ later	18.5
50″ "	10.	100″ "	no change

June 17, 1912, ocular drainage test:

O. P. O. D.	16.	O. P. O. S.	22.
13″ later	13.5	120″ later	no change
65″ "	11.5		
65″ "	10.		

The left eye shows right along lower capacity for drainage and a higher intraocular pressure than the right eye.

Remarks: This patient is interesting on account of the difficulty experienced in deciding the presence or absence of glaucoma in the left eye. She gave a history suggestive of prodromal glaucoma: occasional pains in the left eye, dimness of vision, the seeing of various colors around the gas flame. The field of vision was about 20 degrees concentrically contracted in both eyes, but more limited nasally on the left eye. Both disks were deeply excavated (physiologic excavation?). On the other hand she was markedly neurasthenic and too much importance could not be attached to her complaints. Her vision was good and could be improved to $\frac{20}{30}$ plus with glasses. The intraocular pressure was within the normal limits in both eyes. The opinions of other two ophthalmologists (A and B) of very wide experience was respectively, A: "No glaucoma"; B: "Glaucoma."

The careful study of the behavior of the intraocular pressure in the eyes of this patient and the repeated measurement of the ocular drainage, made the suspicion of prodromal glaucoma very plausible. The reasons are:

A—The intraocular pressure of the two eyes was always found to be different. Those who have made careful examinations of the intraocular pressure in a large number of cases have gained the knowledge that the intraocular pressure is usually the same in both eyes. K. Heilbrun (*Arch. f. Ophth.*, vol. lxxix., 1911), reviewing the findings of Schiötz, Stock, Langenhan, Marple, and Wegner, and his own experience, says, that the i. o. pr. is generally equal in both eyes. He could find only occasional small differences in intraocular pressure of 1–2*mm* Hg., which is within the possibilities of a slight error. Wegner alone found a difference of 8*mm* Hg. in a pair of normal eyes. In the case of Wegner, however, I could not regard both eyes as normal, because one of them had a o. p. of 30, which is above the normal limit.

B—The ocular drainage test in our patient showed that while the intraocular pressure of the right eye presented a certain readiness to recede when the tonometer was steadily applied, the intraocular pressure of the left eye, under the

same test, did not decrease at all, at times, or it decreased at a much slower rate.

The mere fact that the intraocular pressure of an eye is within "normal" limits cannot be considered as conclusive, in my opinion, with regard to the question whether that amount of intraocular pressure is normal. This can be determined only by a comparison of the intraocular pressure and of the draining test of one eye with that of the other.

Another patient in a class by himself is the following:

PATIENT 12. M. L., 38 years old. Simple glaucoma O. S. Coloboma iridis artefactum.

June 1, 1912. O. p. O. D. 50.5*mm* Hg. O.S. 30.5.

June 2, 1912, has been using, for 24 hours, pilocarpin 2% solution, four times daily in each eye. O. p. O. D. 36.5. O. p. O. S. 30.5. We instilled into each eye one drop of a 2% solution of pilocarpin every fifteen minutes for one hour, then we measured the o. p. O. D. 42.5, O. S. 22.

June 14, 1912 has been using pilocarpin 2%, four to five times daily in each eye.

Ocular drainage test:

O. D.	60.	O. S.	26.
50" later	51.	22" later	22.
120" "	no change	120" "	no change

June 17, 1912. Patient has been using pilocarpin drops almost every hour during the daytime.

Ocular test:

O. D.	27.	O. S.	22.
120" later	no change	32" later	18.5
		72" "	16.
		15" "	no change

Remarks: The myotic has reduced the intraocular pressure in the right eye from 60.5 to 27 and the patient assured me that in three years he has not seen as clearly as now. The ocular drainage however was not so greatly influenced. In the left eye with the iridectomy, the myotic reduced the o. p. and improved the rate of drainage. The right eye, as we see, responds readily to the myotics by a reduction of the intraocular pressure, but by no improvement of the drainage capacity.

Of the three groups of patients with glaucoma, the third one is the most interesting. The measurement of intraocular

pressure and ocular drainage is important mostly for the establishment of a diagnosis in suspicious cases of glaucoma. In this group of cases in which the measurement of acuity and field of vision, as well as the ophthalmoscopic examination, could not advance the diagnosis even one step further, the method of measuring the rate of ocular drainage is perfectly welcome if it can help throw any light on the condition of these eyes. It is interesting to note that while the o. p. in these eyes with suspicious glaucoma is within normal limits, the measurement of ocular drainage shows a reduced rate.

SUMMARY

The degree of intraocular pressure depends in a great measure on the integrity of the draining system of the eye, and the examination of the rate of ocular drainage is of more importance than the simple measurement of the intraocular pressure. The rate of ocular drainage can be measured by the aid of the Schiötz tonometer in animal and human eyes with a certain amount of accuracy.

Normal eyes have an ocular pressure and a rate of ocular drainage varying within relatively wide limits; glaucomatous eyes have some impairment in the function of the draining system. It is safe to suppose that in incipient or prodromal glaucoma, the draining system may be undergoing slight alterations which, though too small to raise the intraocular pressure above the upper limit of the normal, yet sufficient to be discernible by the measurement of the rate of ocular drainage. There are patients with eyes in a condition of latent glaucoma in which the intraocular pressure is within normal limits—about 15 to 26*mm* Hg. The diagnosis in such cases can be cleared up to a certain extent by the measurement of the rate of ocular drainage.

UNUSUAL DURATION OF MENTAL SYMPTOMS IN A CASE OF ATROPINE POISONING.

By CHARLES W. BURR, M.D., Professor of Mental Diseases, University of Pennsylvania, Philadelphia Pa.

THE point of interest in the following case is the long duration of mental symptoms, six weeks, in a patient who had been using atropine in the eyes. To save space, and because they were in no way unusual, I shall omit a description of the physical symptoms of atropism she exhibited.

Slight cases of atropine poisoning may present no psychical symptoms though, as a rule, there is a little mental confusion. On the other hand, an amount of the drug which, in the average man, does not produce any untoward effect may, in susceptible people, cause visual hallucinations or illusions of the unreality of which the patient is completely aware. I have seen one case in which the visual hallucinations were experienced only in the predormitium. It occurred in a man who had been ordered small doses of atropine twice daily, by mouth, for a nervous affection. The first night after taking the drug he began to see, while half awake, a spectral human head which changed from moment to moment. He realized its unsubstantiality and it soon passed away and sleep came on. It returned at the same time for several nights and, as he was beginning to worry somewhat, I stopped the drug though no other symptoms had appeared. The hallucination immediately ceased to trouble him. He had never had such an experience before. The only physical symptom he had was slight dryness of the throat coming on as I remember, though my memory is not entirely

accurate about this, about half an hour after taking the drug and lasting about an hour. I suspect that all drugs which produce hallucinations act more promptly and, if one may say so, more easily, on the half-sleeping brain than on one wholly awake. Theoretically this ought to be true; the unemployed brain, and even in the most unintellectual the brain is doing some kind of work (consciously or subconsciously, or perhaps wholly unconsciously) continuously in waking hours, ought to be more susceptible to drug stimuli acting on the centres of the special senses than one employed. My patient could, by thinking of something else, make the vision disappear, but the act of thinking at the same time made him awake completely.

In cases of serious poisoning by atropine the mental symptoms are very marked and characteristic. The picture is the same whether belladonna or its alkaloid has been taken and no matter what the method of administration. The symptoms come on, when once they appear, very rapidly. There is delirium, in severe cases, of the wildest kind, visual and auditory hallucinations and, as a rule, in the fatal cases, convulsions and coma. There is some dispute as to the emotional tone most commonly present in the delirium, some authorities stating that more patients are woful and grief-stricken, others that extreme joy and exhilaration are more frequently seen. The characteristic visual hallucinations are of animals, especially cats, dogs, rabbits, butterflies, and such small fry. Sometimes disgusting, crawling things are seen, but not commonly. Spectres of people, though not infrequent, seem, as a rule, not to be attended to by the patient as closely as those of animals. There may also be seen spectral inanimate objects, but they attract the attention of the patient but little. There is a difference of opinion as to the effect of the drug on the sexual feelings. Some authors speak of marked eroticism, others deny its occurrence, and still others ignore it entirely. All are agreed that priapism is frequent whether with or without emotion. It is possible that some reporters have assumed the existence of eroticism because of the presence of priapism. This is an unjustifiable assumption since, in many disorders, the latter condition is present in the absence of the former. Wakefulness is common.

In the great majority of cases death or recovery occurs within twenty hours. In patients who recover, the symptoms often pass off in a few hours.

The history of the case follows:—M. J., a 52-year-old woman, was admitted to the Philadelphia General Hospital (Blockley) in a state of great excitement and exhilaration and showing visual and auditory hallucinations. Her family physician told me that nine weeks before admission she had been seized with iritis for which instillations of three drops of the standard atropine solution every three hours had been ordered. She continued to use the drops, on her own responsibility, till the day before admission to the hospital, when she suddenly became excited and hallucinatory. His statement was positive that the symptoms came on suddenly and attained their height at once. Her previous medical history was good. She never used alcohol, had never had any mental disorder, and was never even nervous. She was an industrious and thrifty working woman, of a commonplace unemotional type, and not, he assured me, likely to allow her imagination to run away with her: she did not have enough imagination for that.

I examined her two days after her admission. She was a thin, poorly nourished, elderly woman. The excitement had somewhat passed off but she was over-content, almost hilarious, and had no realization of being mentally ill. For twenty-four hours after admission it had been necessary continuously to restrain her physically on account of her extreme restlessness, but need of this had passed off by the time of my visit except that for several nights she had to be fastened in bed as otherwise she would have bothered the other patients. She did not wish to harm them, but would not let them alone. As happens quite frequently in excited patients, physical restraint caused comparative mental quiet without any of the evil effects that often follow the administration of sedatives in sufficient dose to be effective. Though she was almost totally blind she did not realize it. She had both visual and auditory hallucinations. She was not annoyed by them, had no idea of their incongruity, and believed them to be real. She saw people bringing tubs into the room and pulling the furniture down and heard them talking among themselves. They never spoke to her, nor of her, nor was she in any way bothered by their talk. Indeed she paid little attention to what they said. She was very much more impressed by, and interested in, the animals she saw. She constantly saw cats and dogs and rabbits. Rabbits crawled over her bed, cats and dogs played and

fought. " They had taken possession of her house." She liked to play with them and with hands really empty fondled them. At this time she had no realization that she was in a hospital but thought she was in her own home.

Several days later she became mentally quiet enough to make it possible to hold her attention for a short time, and she would then answer correctly questions about her early life and even about the first few days of her eye trouble, but she was entirely confused concerning events later than about seven weeks before her admission. There was, in other words, failure of memory for events immediately preceding the onset of the symptoms, mental confusion, and loss of power to fix attention. She remembered, for example, that she had put drops in her eyes every three hours for several days, but she could not remember when, nor what the drops were for. It was not possible for her to talk responsively for any length of time. Soon her power of attention would fail and she would return to her talk about the dogs and cats. She paid no attention to her dimness of vision and did not regard it as at all important that she could not clearly see the physician she was talking with and that the room looked dark to her. It was not at all strange to her that though she saw the animals clearly all else was blurred and indistinct. The hallucinations continued both night and day. So far as I could tell, they continued present with the eyes closed. After the first few nights she slept well but even during convalescence the hallucinations were most marked in the evening. She was entirely indifferent to her actual surroundings and careless and unconcerned about not seeing her own people.

After she had been in the hospital a few days she began to have falsifications of memory and to "fabricate," or rather these symptoms became noticeable; during the first few days the delirium was too great, the flight of ideas too rapid, for her to fix attention on any one thing long enough to fabricate, though potentiality to fabrication was probably present from the start. Later it became kinetic. She then recognized me as an old friend (I had never seen her) and talked about visits I had paid her. Though really in bed all the time, she told a tale of having been out walking the day before and of having met some soldiers. Some days later, when most of the time she realized she was in a hospital, she told, speaking in a very matter-of-fact way and evidently believing it, the following story: While at home some one put her on the fire escape. (Her home is not fitted with a fire escape.) She went down to the street and walked about all night. Next day she met a couple of officers who after some harmless adventures took her to

the hospital and when she got there she found the bed moving around without any one having their hands on it. The whole story, told here in briefest outline, was imaginary, and was related in a quiet matter-of-fact and yet interesting way, as if it were an every-day experience. When she regained her normal mental state, her power of imagining was so small that she could not have invented such a story. As time passed, the hallucinations became less vivid and less constant, and after she had been in the hospital about two weeks they recurred only now and again daily for a few minutes at a time. Her falsification of memory and "fabrications" became at first more marked as the hallucinations decreased in intensity and later themselves slowly ceased. Excitability and hilarity varied a great deal from day to day but the tendency was to become quieter and quieter. The hallucinations and the other mental symptoms did not entirely clear up till six weeks after her admission and after she had been home some time. She had neither convulsions nor a period of coma during her illness. There was no sexual element in her delirium.

Alcohol occasionally produces the clinical picture described above, but it can be excluded in this case with certainty. Syphilis too might possibly be suspected, but she not only had a negative history, given by a physician who knew her well, but showed no physical signs of present or previous infection. Her vascular and renal systems were in good condition. Had the mental disturbance lasted only a day or two, the picture was so characteristic that no one would have had the slightest doubt about the diagnosis. That she was poisoned by atropine the physical symptoms proved beyond doubt. After considering the whole matter I am forced, notwithstanding the prolonged duration of the illness, to the diagnosis of pure atropine poisoning without any other contributing factor.

REPORT OF A CASE OF CONICAL CORNEA SUCCESSFULLY TREATED BY THE ACTUAL CAUTERY.[1]

By Dr. WM. CAMPBELL POSEY, Philadelphia.

MISS G., *æt.* 25, was referred to the writer by Dr. Wm. F. Weitzel, of Indiana, Pa., in March last, on account of poor vision occasioned by the presence of conical cornea in both eyes. As is usual, there was nothing in the patient's history to throw any light upon the etiological factors which had operated to induce the condition. The vision in both eyes was apparently good in early childhood, but had steadily deteriorated as the patient grew older. Examination showed a conicity of both corneæ, the deformity being, however, much more pronounced in the left eye. Both corneæ were clear and the apices of the cones were situated a little below the pupillary centre in each eye and somewhat to the temporal side. The eye-grounds were normal. With -1.25 D. $+6$ cyl. ax. 160°, vision in the right eye equalled $\frac{5}{12}$, but it was impossible to improve the vision in the left eye more than to $\frac{3}{100}$, and no glass enabled the patient to see even the largest type on the reading card. As the vision in each eye had been steadily failing and it was manifest that that of the right eye would soon be as bad as that in the left, operative procedure was advised, and the consent of the patient being obtained, she was accordingly admitted to the Howard Hospital.

Following his convictions regarding the best means of proceeding in such cases, the reason for which will be presently given, after thorough cocainization of the left eye, the writer cautiously but thoroughly cauterized the temporal base of the cone, the apex being but slightly touched with the hot metal. The area cauterized comprised about one-sixth of the entire structure of the cornea and was triangular in form, the apex corresponding to the summit of the cone, the base to a position in the cornea about *2mm* distant

[1] Read before the Section on Ophthalmology of the College of Physicians of Philadelphia on December 19, 1913.

from the limbus and parallel with it. A large strabismus hook heated over an alcohol flame was employed as the cauterizing instrument, its tip being applied at a red heat. After thorough atropinization, the conjunctival cul-de-sac was filled with iodoform ointment and a firm compress bandage applied. Healing was uneventful but protracted, though cicatrization was favored by the presence of a number of blood-vessels which invaded the cauterized area from the neighboring limbus. At the end of six weeks, the reaction occasioned by the operation had subsided sufficiently to permit the patient to return home, without, however, an attempt being made to order a correcting glass for the operated eye.

After a period of seven months, the patient returned, highly pleased with the result of the operation, averring that she could now read ordinary newspaper type and that her vision, even without a glass, was excellent. When tested, uncorrected vision in the left eye was found to equal $\frac{5}{\frac{1}{2}}$, and with $-1. + 3.5$ cyl. ax. $35° = \frac{5}{\frac{1}{2}}$. Type 0.75 D. was read without a glass, and with the above correction, type 0.50 D. Vision in the unoperated eye, however had markedly fallen, being less than $\frac{5}{100}$. The operated eye, was entirely quiet, though somewhat disfigured by the presence of the scar tissue. At least a half of the pupillary area of the cornea was uninvolved by the opacity, however, and as the edge of the macula was dense and defined from the surrounding corneal tissue by a sharp line of demarcation there was no disturbance of vision by aberrant refraction. Tattooing was advised, but not insisted upon, the operator being as well satisfied as the patient with the visual results and being desirous, as was she, of subjecting the right eye, to a similar operation. This was accordingly done, in precisely the same manner as that just described; the healing process in this eye was less protracted and the patient was permitted to return to her home at the end of three weeks. A recent letter states that the result of the operation upon the right eye is equally as good as upon the left. It is the intention of the patient to comé to Philadelphia some time later in the winter, when the writer will endeavor to present her before the Section. Tattooing of the left cornea will probably be performed at that time also.

In most instances, vision in conical cornea can be satisfactorily improved by high cylindrical lenses; in some cases, however, as in that which has just been cited, the conicity and distortion of the cornea is so great that lenses are useless, and operation is demanded.

The surgical measures which have been instituted by operators from time to time, for the relief of advanced cases of conical cornea, have been varied and may be divided into two classes: (1) those which aim to improve vision by either an iridectomy, iridodesis, and iridotomy without attempting to change the shape of the cone; and (2) those which have been designed to flatten the conicity of the cornea, either by excision of circular areas and ellipses of the membrane either in conjunction with or without cauterization, or by cauterization alone.

The results attendant upon the creation of an artificial pupil without previous operation to flatten the conicity of the cornea, have been so unsatisfactory that they are but in little favor at the present time, while the removal of a segment of the cornea is attended with such danger, not only on account of the likelihood of injury to the lens by the instrument employed in the excision, but also by reason of the complications arising in the healing from the thinness and malnutrition of the cornea, that cautious surgeons regard this method of procedure also with considerable apprehension regarding the ultimate result.

Most operators, therefore, favor some form of cauterization, aiming to substitute by this method a resistant and flattened cicatrix for the attenuated apex of the cone. According to both Oliver and Nance in Wood's *System of Ophthalmic Operations*, von Graefe was the first to employ this plan of procedure, cauterizing the conic apex, which he had first scarified with a cataract knife, by repeated applications of nitrate of silver. Care was taken that the cornea was not perforated, though in some cases the aqueous humor was evacuated by a paracentesis, made every few days over a period of about two weeks. A bandage was applied until the healing process was well advanced. At times the resulting scar covered the pupil and iridectomy was necessary.

In later years, the cauterizing agent has usually been the galvano-cautery, and operators vary in their methods only in the extent of the area cauterized and in their treatment of the contents of the anterior chamber, some perforating into the chamber with the cautery at the time of the cauterization, while others have tapped the chamber by paracentesis some

days later. There are those, on the other hand, who see no necessity for emptying the chamber, thinking that this procedure needlessly complicates without aiding in the subsequent flattening of the scar tissue.

After a careful consideration of the views of different operators and from the study of several of his own cases, as well as others of his colleagues, notably of one most successful case reported by Dr. P. N. K. Schwenk, the writer believes that success from cauterization lies in replacing the greater part of the cone by a mass of firm connective tissue, which should be so placed that the traction exerted by the scar in the process of healing is sufficient to resist the intraocular pressure, while its density should be great enough to prevent rays of light from penetrating it, thereby reducing aberrant refraction. As the apex of the cone seldom occupies the pupillary centre, the resultant scar need not entirely block the pupil, so that iridectomy, is as a rule, unnecessary. As Elschnig has pointed out, prompt vascularization of the cauterized mass is most desirable, and the writer follows his advice to connect the wound in the cornea with the conjunctiva by carrying the cauterization almost to the limbus. The writer also believes in the graduated application of the cautery as advocated by Sir A. Critchett and H. Knapp, although like Sgrosso he cauterizes the temporal wall of the cone as deeply as possible, the apex being burned but slightly. A firm compress bandage is essential to support and flatten the cornea, and should be maintained for several weeks (after the operation). It should, however, be removed twice daily to permit of flushing the eye with boracic acid lotion, for the instillation of atropine, and for a copious enunction with iodoform ointment.

Subsequent tattooing of the corneal scar for cosmetic purposes is, of course, optional with the patient. Should the scar tissue, however, be not sufficiently dense in places, and should the rays of light pass through it, the implantation of a thick layer of pigment into the scar, thereby absorbing such rays, should be of value in still further improving visual acuity.

ILLUSTRATING DR. VERHOEFF'S ARTICLE ON THE EFFECT OF CHRONIC GLAUCOMA ON THE CENTRAL RETINAL VESSELS.

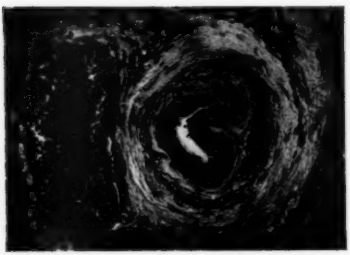

FIG. 1. Endarteritis of central retinal artery in secondary glaucoma, showing infolding of actively proliferating intima.

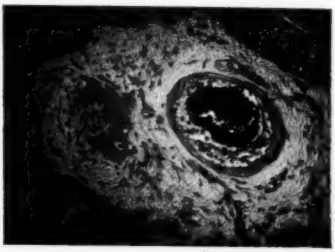

FIG. 3. Endovasculitis of central retinal vessels in secondary glaucoma. The vein on the left shows proliferation of its intima with papillary projections into the lumen. The artery shows fatty endothelial cells in its intima.

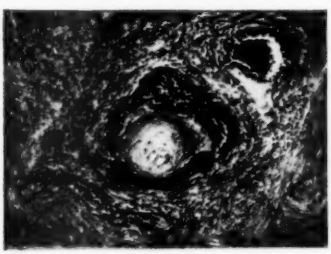

FIG. 2. Dissecting aneurism of central retinal artery. There is a central tube with obliterated lumen, surrounded by a space filled with blood. In subsequent sections a branch is seen entering this space.

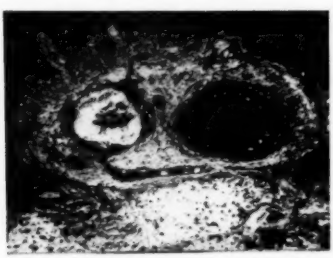

FIG. 4. The central retinal vein, on the left, shows an unusual form of dissecting aneurism. There is a central hyaline tube with obliterated lumen, surrounded by a space filled with blood from a branch entering below. Note similarity to artery in Fig. 2.

THE EFFECT OF CHRONIC GLAUCOMA ON THE CENTRAL RETINAL VESSELS.

By F. H. VERHOEFF, A.M., M.D.

From the Massachusetts Charitable Eye and Ear Infirmary.

(*With four illustrations on Text-Plate V.*)

THE effect of chronic glaucoma, either primary or secondary, on the central retinal vessels has not hitherto been determined. In cases of primary glaucoma, it is true, these vessels have frequently been found affected, but here it has been assumed that the vascular changes have been part of a general angio-sclerosis upon which the glaucoma itself was perhaps dependent. On the other hand, the effect of secondary glaucoma on the central vessels has not previously been investigated, although obviously the results obtained would apply also to primary glaucoma. For in a series of properly selected cases of secondary glaucoma, it is evident that whatever changes are found in the central vessels must, in general, be dependent directly or indirectly on the increased intraocular pressure. It is the purpose of this paper briefly to summarize the findings in such a series of cases.

The material for this investigation consisted of all available eyes in this laboratory from cases of secondary glaucoma due to lesions of the anterior portion of the globe. Many eyes were not available because the optic nerves had already been sectioned longitudinally. Only cases with definite cupping of the optic disk were included, so as to allow no doubt as to the presence of glaucoma, and care was taken to exclude all cases in which the clinical records indicated the least probability of primary glaucoma in either eye. Cases with retinal hemorrhages were not excluded, but of these there

were only three. So far as concerned changes in the central retinal vessels, these cases comprised an unselected series.

The fixation was in 10% formalin followed by a saturated solution of potassium bichromate for twenty-four hours. In every case serial celloidin cross-sections were made through the region of the lamina cribrosa by the method described by me elsewhere,[1] and stained, usually by my elastic tissue stain,[2] which is also an excellent nuclear stain.

The total number of cases was 39. The causes of the glaucoma were: occlusio pupillæ, 8 cases; anterior synechiæ, 19 cases (corneal ulcer 6, injury 13); anterior staphyloma, 8 cases; cataract extraction with incarceration of lens capsule in wound, 2 cases; serous iritis, 1 case; small sarcoma of the ciliary body with metastases in the filtration angle, 1 case.[3] The ages were: 1 to 10 years, 5 cases; 10 to 30 years, 7 cases; 30 to 50 years, 19 cases; 50 to 70 years, 8 cases. The durations of the cases, estimated by the time elapsed since the corneal ulcer, injury, etc., were: less than 2 years, 13 cases; between 2 and 10 years, 17 cases; between 10 and 20 years, 7 cases; over 20 years, 2 cases.

Microscopic examination of the central vessels in the above cases showed that in every case one or both of the central vessels were affected with endovasculitis, in the region of the lamina cribrosa. The vein was the more affected in 19, the artery in 15 cases, while the two vessels were equally affected in 6 cases. In 7 cases the vein, and in 3 cases the artery, was normal. In 8 cases there was complete, and in 5 cases almost complete, obstruction of the central vein. In 4 cases there was complete, and in 2 cases almost complete, obstruction of the central artery. In 2 cases there was complete or almost complete obstruction of both vessels. In general, therefore, the vein was the more greatly affected of the two central vessels. In 8 cases it is noteworthy that the central vessels were only slightly affected in spite of the fact that in each the glaucoma was in an advanced stage with deep cupping of the disk.

[1] "Obstruction of the Central Retinal Vein." ARCHIVES OF OPHTHALMOLOGY, 1907, xxxvi., 32.

[2] "An Improved Differential Elastic Tissue Stain." *Journal American Medical Association*, 1911, lvi., 1326.

[3] Reported in full in *Ophthalmology*, July, 1908.

Retinal hemorrhages were present in only three cases, each showing complete obstruction of the central vein. In none were they as abundant as is usual after primary obstruction of this vessel.

In the first of these cases (2—2005) there was only one punctate hemorrhage, near the macula, advanced atrophy and moderate œdema of the retina. The vein showed a thick hyaline wall and its lumen was occluded by active cellular subendothelial proliferation. The lumen of the artery was reduced to about half its normal size by endarteritis. The patient, aged 43 years, had received a perforating wound of the cornea and lens fifteen years before, the secondary glaucoma evidently being due to incarceration of the lens capsule in the wound following extraction of the traumatic cataract.

In the second case (2—1217) there were a few punctate hemorrhages near the disk and macula. The optic disk showed only moderate cupping with cavernous atrophy. The retina showed only slight atrophy, many multipolar ganglion cells still being recognizable, and slight edema. The vein was completely occluded in only a few sections, by active subendothelial proliferation. The artery was only slightly affected. In this case there was a leucoma adherens and traumatic cataract due to a perforating injury with a stick of wood one year previous to enucleation. The patient was aged 60 years.

In the third case (2—2014) punctate retinal hemorrhages were fairly numerous in the region of the equator and there was slight retinal œdema. Multipolar ganglion cells were still present in the macula, and the optic disk was only moderately cupped. The vein showed a hyaline wall and the lumen completely occluded by a relatively active subendothelial proliferation. The artery was only moderately affected but showed a dissecting aneurism. The patient, aged 43 years, two years preceding the enucleation had an attack of ulcerative keratitis following malaria, with Saemisch operation and resulting leucoma adherens.

In four cases there were expulsive subchoroidal hemorrhages occurring spontaneously in three and after optical iridectomy in one. In one of these the central vein, and in two the artery, was almost completely obstructed.

Age was apparently not an important factor in determining the degree of endovasculitis or its preponderance in the vein or artery. This seemed to be true also of the duration, but

here, of course, the duration of the effective increase of tension may often have been less than that of the causal lesion. About the only definite conclusion that can be drawn in regard to age and duration is that in young individuals secondary glaucoma never produces marked changes in the central vessels in as short a time as is sometimes seen in old individuals.

As regards the character of the changes in the central vessels, essentially the same types were met as those that have been described in the literature, and that I have myself observed as occurring in hemorrhagic glaucoma. In no case was there any evidence of thrombosis, though in some cases without the aid of serial sections the changes might have been mistaken for thrombosis with organization. In the artery the simplest change was a subendothelial proliferation of spindle cells, usually more marked on one side, sometimes associated with the formatien of collagen and elastic tissue. At intervals the elastic tissue became condensed, so that in some cases as many as three or four concentric elastic membranes were present. This would seem to indicate that the process was intermittent. The cells immediately about the lumen almost always showed the most active proliferation so that the lumen became distorted. This was especially well seen in one case (2—1866) where the lumen was obliterated by an infolding of the actively proliferated intima (Fig. 1). In some cases, probably where the process was slow, the lumen was completely occluded by elastic tissue with almost no cells. More often, however, a tendency was manifest for the new tissue to undergo necrosis in a middle zone immediately internal to the old internal elastica. This degenerated zone was transparent and often contained fatty endothelial cells. No doubt it was due to its nourishment being cut off by the tissue around the lumen. As a result of this process an inner tube was often separated off, the space around which in five cases was distended with blood, thus forming a dissecting aneurism (Fig. 2). In two of these cases the space was filled with blood from branches of the artery, but in the others no branches were found in this region, so that it was probable that the degenerated intima was undermined by the central blood stream. The media was not affected until late, when it showed atrophy and ultimately disappeared. Spindle

cells around the lumen sometimes closely resembled a new-formed media.

The changes in the vein were analogous to those in the artery. The primary change was evidently subendothelial cellular proliferation with formation of connective tissue. New elastic tissue was also often formed, but this was always in finer fibrils and less abundant than in the arteries. When the process was evidently active, new cells were abundant and the connective tissue slight in amount; when slow, few cells were present and the new tissue was hyaline or fibrous. When very active, the cells were very small and might have been mistaken for lymphocytes. The new-formed connective tissue was sometimes not easily differentiated from the old adventitia, so that an appearance was produced of a normal vein with a very small lumen. In some cases the vein was almost completely occluded in this way, and the remaining small lumen obliterated by endothelial proliferation. In other cases the vein wall was hyaline, so that the vein somewhat resembled an iris vessel. In most cases the vein wall was unevenly involved and irregular or partly collapsed, in this way the size of the lumen being reduced. In some cases the proliferated intima projected into the lumen in a papillary manner (Fig. 3). Fatty degeneration was not evident, though no doubt suitable fat stains would have shown more or less of it. In four cases a condition not hitherto recognized was noted. This was the encroachment of a bundle of neuroglia upon the wall of the vein. The wall of the vein, perhaps as a result of this, was collapsed and the lumen reduced to a narrow slit. Without the aid of complete serial sections this appearance might well have been mistaken for a large vein filled with actively proliferating endothelial cells.

Just as in the case of the artery, degeneration often occurred around the new intima and the space so formed sometimes filled with blood. Dissecting aneurisms were thus formed in six cases, in five of which the source of the blood was found to be a branch or collateral. In one case there was an inner tube, surrounded by blood, such as just described as occurring in the arteries, the wall of which was necrotic and its lumen closed by endothelial proliferation. Fig. 4 shows a branch entering the space around it. So far as I know, this condition

has not hitherto been described. Another form of dissecting aneurism was produced in an analogous way but presented a different picture, the lumen of the vein being divided into several compartments by septa. This condition has been noted several times by other observers, in cases of hemorrhagic glaucoma, notably by Coats and Harms, who regarded it as due to canalization of a thrombus. In a previous case,[1] by means of a complete series of sections I was able to show that it really was a dissecting aneurism, the septa being due to the undermining of the degenerated intima by the blood from a branch, and the bulging into the lumen of the intima thereby produced. This explanation was fully confirmed by the findings in the present series of cases.

The explanation of why the central retinal vessels should be so markedly affected in secondary glaucoma is not obvious. Three factors at least, however, must come into consideration; namely, the direct action of the increased intraocular pressure on the retinal vascular system, the action on the central vessels of toxic substances resulting from the relative stagnation of the intraocular fluids, and the traction on the vessels produced by the receding lamina cribrosa. The first of these seems to me to be the most important factor of the three. It seems entirely probable that the increased intraocular pressure, by compressing the retinal vessels and thus raising the pressure in the central artery, would cause a so-called compensatory proliferation of its intima. The explanation of the still more frequent changes in the central vein, however, is not so clear, for the pressure in the vein would be diminished instead of increased. Possibly this diminished pressure, especially if associated with collapse of the vein wall, would of itself lead to intimal proliferation. Another possible explanation of the latter is that it is due to the excess of carbon dioxide or lack of oxygen in the vein resulting from the impeded circulation in the retina. Where the artery was also affected this explanation would apply with still greater force.

The almost complete absence of retinal hemorrhages in this series of cases is noteworthy. Thus out of fifteen cases of complete or almost complete obstruction of the central

[1] "Obstruction of the Central Retinal Vein," ARCHIVES OF OPHTHALMOLOGY, 1907, xxxvi., 32.

vein, in only one case were they present in any number. In two other cases there were a few small hemorrhages, but it was doubtful if they were the result of the obstruction of the central vein. This infrequency of retinal hemorrhages was probably due in some cases to the marked involvement of the artery, and in others to the slowness of the process allowing the establishment of adequate collateral circulation in the nerve. Œdema of the retina for similar reasons was not marked in any case.

It is obvious that in chronic primary glaucoma the central vessels must be subjected to the same influences as in secondary glaucoma, and hence must likewise undergo proliferative and degenerative changes. This suggests that the diminution in vision in chronic glaucoma may be largely due to partial obstruction of the central vessels. There can be no doubt that in the absence of other causes complete blindness ultimately would be produced in this way. Nevertheless it is not probable that endovasculitis of the central vessels is a frequent or important factor in the loss of vision in glaucoma. This is indicated by the fact that, with two exceptions, changes in the central vessels sufficient to cause impairment of retinal function occurred in only advanced cases of secondary glaucoma in which there was already atrophy of the optic nerve and retina, and also by the fact that in eight cases, equally far advanced, the central vessels showed only moderate changes. In other words, it is probable, generally speaking, that loss of vision in chronic glaucoma is already practically complete before marked obstruction of the central vessels occurs. It is possible, however, that where a tendency to general angio-sclerosis already existed, chronic glaucoma would produce obstruction of the central vessels more quickly.

The fact, established by these cases, that chronic glaucoma leads ultimately to complete obstruction of the central vessels brings up again the question previously raised by me whether or not many cases reported in the literature as instances of primary obstruction of one or the other central vessel are really such. It is certain that some of them are cases of primary glaucoma, but just which ones it is difficult to determine. This question, which is of some importance from the standpoint of diagnosis and treatment, will be discussed more

fully in a later communication dealing with primary and hemorrhagic glaucoma.

CONCLUSIONS

Secondary glaucoma always produces, sooner or later, endovasculitis of the central retinal vessels, leading ultimately to complete obstruction of both. The same variety of changes are seen as in cases of primary obstruction of these vessels. Thrombosis does not occur. Dissecting aneurisms not infrequently occur in both the vein and artery, producing complicated appearances which have been mistaken for canalized thrombi.

Obstruction of the central vein due to secondary glaucoma, probably owing to its slow development, seldom if ever produces the intense hemorrhagic retinitis often seen after primary obstruction of this vessel.

In chronic primary glaucoma, endovasculitis of the central retinal vessels, where not coincidental, is the result, not the cause, of the increased intraocular pressure. In the majority of cases it probably does not reach a sufficiently high degree to impair vision until the latter is already lost from other causes.

THE OPTIC DISK IN PURULENT OTITIC DISEASE AND ITS COMPLICATIONS.[1]

By Dr. E. GRUENING, New York.

CHANGES in the appearance of the optic disks occur in purulent middle-ear disease and its complications. In 1871 Clifford Allbutt, now Sir Clifford Allbutt, wrote a book entitled, *On the Use of the Ophthalmoscope in Diseases of the Nervous System, etc.* It is a remarkable book, as modern today as it was more than forty years ago. Allbutt's observations and interpretations of the appearance of the optic disk in health and in disease are based chiefly upon the teachings of Albrecht von Graefe. What interests and concerns us particularly, is the concise and exact differentiation of optic neuritis and choked disk. These two distinct pathologic conditions of the optic disks occur in the course of purulent otitic disease and its complications; and their precise differentiation has an important bearing upon diagnosis, prognosis, and therapy of the disease.

In 1879 Charles J. Kipp, of Newark, N. J., and in 1881 Zaufal of Prague urged the use of the ophthalmoscope in all stages of suppurative acute and chronic ear disease. Zaufal considered the examination of the diseased ear incomplete without the ophthalmoscopic finding.

In our present-day otologic literature the condition of the optic disk is but casually referred to, and it seems that its diagnostic significance is more lightly regarded than formerly. At times the statement is made that an optic neuritis has developed into a choked disk, and this implies that choked disk is a more advanced stage of optic neuritis. This is an erroneous view.

[1] Read before International Otological Congress, Boston, 1912.

Choked disk has an initial, an intermediate, and an advanced stage, every one of which is the ophthalmoscopically visible expression of increased intracranial pressure. The German term "Stauungspapille," introduced by Albrecht von Graefe, expresses the various phases of local engorgement more precisely than the English term "choked disk," which applies to the most marked stage alone. It is as easy to diagnosticate the pronounced choked disk as it is difficult to recognize the earliest stage. The true value of such an early recognition, however, lies in the fact that the diseases in which choked disk occurs are amenable to treatment. Optic neuritis in otitic complications, on the other hand, is a frequent concomitant of purulent meningitis, and in that disease our present-day therapeutic measures have hitherto proven futile.

In acute purulent meningitis due to otitic or other causes, the outlines of the optic disk are often blurred, and here there is a true inflammation of the optic nerve and its disk. Though there may be an increase of intracranial pressure due to an excess of cerebro-spinal fluid, there is no choked disk because, early in the course of the disease, the intervaginal space is sealed by exudative material, and the serum does not enter the lymph spaces of the optic nerve and the perilymphatic sheaths of the optic and retinal vessels.

How can the choking of the disks, which lie so far from the area of disease, in purulent middle-ear affections be explained? If we turn to text-books on ophthalmology or otology, we do not find in either a succinct and lucid explanation of this inter-relation. It is a known fact that an increased intracranial pressure due to an excess of cerebro-spinal fluid causes choking of the disks. But what causes the excess of cerebro-spinal fluid? Theodor Leber's theory of chemotaxis may furnish the desired clue. The pyogenic germs in the otitic area have a wide sphere of action, and may, by the diffusion of their toxins, induce a change in the walls of distant vessels, allowing a transudation of the serum of the blood.

What is known as meningismus, serous meningitis, and serous labyrinthitis may be caused by an excess of serum without inflammatory products.

Another point of interest is that the degree of swelling may vary in the two disks. As we assume that the hydrostatic

pressure is equal at all points within the brain there must be local structural conditions causing these variations. The recognition of a higher degree of swelling of one disk determined my plan of action in a case of double mastoiditis with symptoms of thrombosis of the lateral sinus. It was doubtful in this case which of the sinuses was involved. As one optic nerve was more swollen than the other, it was assumed that the sinus was involved on that side. This conclusion was verified by the operation.

Further confirmation that we have not to deal with inflammatory action and destruction of nerve tissue in the choking of the disks in otitic cases is supplied, in my personal experience, by the fact that on removal of the purulent area, be this a purulent mastoiditis, an extradural abscess, a sinus thrombosis, or any other complication from which the patient recovers, the optic nerves finally assume their normal appearance, and show no impairment of function.

As to the genesis of choked disk, I wish to mention especially that Schiek's theory[1] explains best the facts which have come under my observation.

[1] "Beiträge zur Kenntniss der Genese der Stauungspapille," Franz Schiek, *Arch. für Ophthalmologie*, Band 78, Heft 1.

THE RODS AS COLOR-PERCEPTIVE ORGANS.

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Translated by Dr. Alfred Braun, New York.

At present the view is prevalent, that the chromatic apparatus of the retina is represented by the cones, and that the rods are color-blind organs, whose function is to intermediate the impressions of weak light-intensities—that is, that they constitute a dark-chamber apparatus.

This theory was first propounded by Max Schultze. Schultze supported his theory upon a series of comparative anatomical studies. Nocturnal animals, such as bats, moles, owls, etc., have scarcely any cones, whereas the rods are exceptionally well developed. For this reason, Schultze thought that the rods constituted an apparatus for seeing in the dark, and as colors do not exist in the dusk, these organs are color-blind. Furthermore, since the color-sense of the human retina is most markedly developed in the centre, and diminishes toward the periphery, and the cones are so arranged, that they are most numerous in the centre, Schultze came to the conclusion that the chromatic apparatus of the eye was formed by the cones.

This theory was then further developed by Parinaud and especially by von Kries and his school, and is at present accepted by most observers.

Some experiments in color physiology, performed by von Wendt and myself, eight years ago, caused us to doubt the truth of the present teaching of the total color-blindness of the rods. I tried then, in several papers, to show that the rods intermediate color-sensations, of short wave-lengths—*i. e.*, violet and blue. As some experiments were recently

published, whose results seem to support this theory, I thought it would be of some interest to set forth the reasons, here, against the von Kries duplicity theory, and in favor of the view that the rods are also chromatic organs.

The most important support of the theory of the complete color-blindness of the rods is, according to von Kries, the appearance of the dim spectrum. As this monochromatic spectrum is colorless, and as von Kries and his school have shown by exhaustive studies that it is the rods which receive the light-impressions from this spectrum, von Kries concluded that these organs intermediate only colorless impressions. Such a conclusion seems, *a priori*, somewhat hasty, especially so if the so-called lack of color of the dim spectrum is examined closely. This statement is not correct, as the dim spectrum has a beautiful bluish tone, as can be very easily demonstrated. Von Kries himself mentions this fact, although only in passing. "In reality," he writes, "there are certain facts, which tend to show that rod-sensations are not really colorless, but somewhat bluish." Yet von Kries has completely neglected this fact, namely that this spectrum is not really colorless, in order to favor his duplicity theory, which demands a complete color-blindness of the rods.

As the theory of the complete color-blindness of the rods depends largely on a series of examinations of the dim spectrum, it seems to me that the fact that this spectrum is really colored, is of considerable importance in deciding the entire question. In support of the duplicity theory, there has been brought forth the fact of so-called complete color-blindness (monochromasia). The characteristic thing in persons who present this anomaly is, that they cannot distinguish between the different colors in the spectrum. The entire spectrum seems to them to consist of a single color. In other words, they are monochromates. What is particularly interesting, is that the division of light in the spectrum is the same for these people as for a normal eye adapted for darkness. From this fact, the conclusion was drawn, that these so-called completely color-blind people see only with the rods, and have assumed that they see no colors whatever, but that everything appears gray.

The logic of this conclusion is, however, by no means bind-

ing. For, even though these people see the spectrum as one color, it does not follow that the impression given by this simple spectrum is that of complete absence of color. On the contrary, it is very probable that these so-called "rod-seers" see the spectrum in the same bluish tone as a normal eye adapted for darkness. The term total color-blindness is therefore not very apt, and should be changed to monochromasia. This anomaly cannot be considered as a support for the theory of the complete color-blindness of the rods.

Max Schultze brought forward, as an argument in favor of this theory that the cones constitute the chromatic apparatus, the fact that the color-sense is most marked in the centre of the retina, and diminishes toward the periphery, which conforms to the arrangement of the cones in the retina. However, Schultze's view in regard to the distribution of the color-sense is only partly correct. It is true only for light of long wave-lengths. The perception of light of short wave-lengths is just the opposite.

In connection with this, some of the circumstances in connection with the color-fields should be touched upon. On account of their varying extent, we are entitled to assume that the color-perceiving apparatuses for red and green lie more in the centre of the retina, while the apparatuses which perceive blue and yellow lie more in the periphery. If we persist in the assumption that the cones only constitute the chromatic apparatus, it is very difficult to explain this variation in the extent of the color-fields. This can be explained only by assuming that color-perception is distributed among various cones, so that one cone perceives only one color (red, green, or violet), or two colors (red-green or yellow-blue). In Oehrvall's laboratory in Upsala, Oerum studied this question, and came to the conclusion that each fundamental color was perceived by a different cone.

Therefore, the red and green perceiving cones are most numerous in the centre, while the violet perceiving cones occur also in the periphery. However neither Oerum's investigations nor the Young–Helmholtz theory give any explanation of the fact, that the field for yellow extends far beyond the limits of the red and green fields. For, since yellow, which, according to the Young–Helmholtz theory, occurs as the result of

the combined action of the red and green perceiving apparatuses the color field for yellow must coincide with that for red and green.

I have tried to test the foundation upon which the Schultze–v. Kries teaching rests, and there is no denying the fact that the foundation is a very weak one. In addition, there are many observations in color physiology which do not agree with this duplicity theory. The most important of these is the fact of the varying sensitiveness of the retina for light of varying wave-lengths, in such a manner that the centre is more readily excitable for light of long wave-lengths than for light of short wave-lengths, while the periphery acts in just the opposite way.

This fact is very well illustrated by the well-known Purkinje's phenomenon. This consists in the fact that the color-perception of light of long wave-lengths disappears before that of short wave-lengths, when the intensity of the light is diminished, and the adaptation of the eye is changed When, for instance, a red and a blue paper appear to have the same light intensity by daylight, with the onset of dusk, the blue appears lighter, and the red darker. As it becomes darker, the red disappears before the blue, and for this reason, as v. Helmholtz says, on a dark night, when no other colors are seen, the blue of the sky can still be distinguished.

This phenomenon has received especial attention from all experimenters, and has been explained in various ways.

Helmholtz was of the opinion that it depended upon objective differences in the light intensity; that the phenomenon was a purely physical one. On the other hand, Hering showed by ingenious experiments that it depended upon the adaptation of the eye. He tried to explain this in harmony with his theory of the specific light-strength of the colors. This theory teaches that each color possesses two valencies, one colored and one colorless, and furthermore, that in light of long wave-lengths, the colorless valency is less than in light of short wave-lengths. Purkinje's phenomenon rests, according to Hering, upon the so-called "receptive capacity" of the two colors. In darkness, the colored valencies disappear, while the colorless persist. As in light of short wave-length (blue) the white valency is stronger than

in light of long wave-length (red), it is easily seen why, in the darkness, the blue color appears lighter than the red.

This is not the place to attempt a criticism of this explanation. Only one point must be brought out, which shows the error of Hering's view, and is, at the same time, of considerable importance for the teaching of the color-blindness of the rods. Hering claims (and according to his theory of valencies he must do so) that the color-perception of red disappears at the same time as that for blue. However, this is not the case. Helmholtz says that the red color disappears earlier, and that when the perception for all other colors has gone, the perception for blue still remains. That this is really so, any one can easily convince himself. For the physiological explanation of Purkinje's phenomenon, the fact that it does not occur in the fovea, is of considerable importance This fact was first observed by Parinaud, and denied by Koster, Tschermak, Hess, and others, and finally proven to be true by v. Kries and Nagel. I have also found that Purkinje's phenomenon is not present in the portion of the retina which contains no rods. Von Kries ascribes the absence of the phenomenon in the fovea to the absence of the rods here.

By proper observance of Purkinje's phenomenon, it can be determined that when the fovea centralis (or in other words the cones) no longer reacts to light of short wave-lengths, the periphery of the retina still perceives such light, and that here there is a distinct sensation of color. This important circumstance has been too little noted. However it proves that this color perception can be intermediated only by the rods.

Purkinje's phenomenon demonstrates in an instructive way the color-perceptive powers of the rods. In support of this theory, I might also mention v. Wendt's and my studies of yellow vision in santonin poisoning. After taking a certain dose of santonin, all white objects appear a light yellowish-green, in daylight. Shadows and dark objects appear reddish violet. If the eye of a person poisoned by santonin looks at a spectrum, the violet color is absent. The eye is, in other words, violet-blind. This is the cause of the yellow vision. All white objects must appear in the color which occurs when the violet color disappears—that is, in its complementary color, yellowish-green.

On closer examination of this so-called yellow vision, one observes that it occurs only in the periphery of the retina. Central vision remains white. A perimetric examination of v. Wendt's and my own santonin poisoned eyes showed that in an area of 8–10° about the fixation point, there was no yellow vision. This fact speaks in favor of the view that santonin affects in some way the formation of visual purple in the retina. This view is supported by Filehnes' investigations, which demonstrated that in santonin-poisoned frogs there were proven to be disturbances in the formation of visual purple.

Consequently we must look for the seat of the violet-blindness in santonin poisoning, in a disturbance of the normal function of the rods, from which we deduce the fact that under normal conditions these organs cannot be color-blind.

A similar yellow vision occurs in icterus. This color-phenomenon was investigated by E. Rose, Hirschberg, and myself. Hirschberg was of the opinion, that it depended upon a yellow coloring of the media of the eye. For various reasons, this cannot be so, and we must, in agreement with E. Rose, look for the disturbance in the retina itself. As I was in the position to observe, the yellow vision during icterus is also limited to the periphery of the retina, while the purple-free region sees white. While this speaks against a simple yellow coloring of the media, it proves at the same time that the seat of the disturbance is in the rods. As we know that the biliary salts are the only material which can dissolve the visual purple from the rods, we are led to the belief that this disturbance is caused in some way by the biliary salts which circulate in the blood of the icteric patient.

Furthermore, in patients with hepatic disease, hemeralopia is not uncommon, a fact which would point to disturbances of the liver function as factors in influencing the rods of the retina.

Hemeralopia itself may also be used as a support for the theory of the color-perception of the rods. It is a well-known fact that hemeralopia is usually accompanied by blue-blindness (violet-blindness?). If hemeralopia depends upon a disturbance of the rods, it is very likely that the disturbances of color-vision which accompany hemeralopia are also caused by the rods.

In 1904, Lundsgaard claimed, as a result of his observations of red vision in hemeralopia, that the visual purple and the rods are concerned either directly or indirectly in the perception of blue.

It is characteristic for hemeralopes, that Purkinje's phenomenon does not occur among them. This speaks for my explanation of this physiological occurrence, and also for the correctness of the assumption that the rods have color-perception.

In regard to the latest investigations, I would like to discuss Hess's interesting researches on the color-perception of day- and night-birds, as these investigations support my theory.

Hess's investigations show the error of Parinaud's teaching of the rôle of the visual purple in hemeralopia. In support of his theory, Parinaud has stated the fact that animals who have no visual purple, for instance the day-birds (chickens, pigeons), are hemeralopes, whereas the nocturnal birds, which have considerable visual purple in the retina, see very well after dusk.

I do not wish to dwell on the investigations of seeing in the dark, but merely want to touch on the results which Hess's work brought out in regard to the color-perception of these birds. They show that day-birds, which have no visual purple, have a very much shortened spectrum in the short-waved portion.

The violet, blue, and bluish-green rays do not appear to be able to excite the retina of these animals. On the other hand, the nocturnal birds, with their profusion of visual purple, see the short-waved portion of the spectrum almost as well as the human eye.

Yet Hess does not connect this fact with the difference in the occurrence of visual purple in these animals, plainly because he looks upon the cones, *a priori*, as the sole chromatic organs. He looks for the cause of this difference in the yellow-colored oil-globules which occur in the inner portions of the cones of these birds. These oil-globules are supposed to absorb the short-waved light, which is thus prevented from reaching the outer portion of the cones. If this were the explanation, we would expect that the yellow-colored oil-droplets are not present in nocturnal birds, as these perceive the short-waved light.

However, this is not the case, as these birds also possess these globules. But, says Hess, in these animals the globules are less intensely colored, hence the difference.

It is likely that the real explanation of the above-mentioned fact is to be found in the unequal presence of visual purple in the day and nocturnal birds. Thus Hess's investigations also support the theory of the color-perception of short-waved light by the rods.

With this view Köllner's newest investigations on the relationship between adaptation for darkness and violet-blindness can be brought into harmony. I wish only to touch upon that here.

I have tried to discuss here the most important facts in color physiology, which disprove the theory of the color-blindness of the rods. Some time ago, Ebbinghaus and Koenig combated the view that the visual purple constituted the violet or blue-perceiving substance, according to Helmholtz's color-theory. As I had the honor to show, so many of the newer observations speak against v. Kries's duplicity theory that, in my opinion, it can no longer be upheld. On the other hand, all of the facts hitherto known, in the study of color, can be brought into harmony with the theory that the rods are the organs which are concerned in the perception of color of short wave-lengths.

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ON THE ETIOLOGY OF PARINAUD'S CONJUNCTIVITIS.

By Dr. KRUSIUS and Dr. CLAUSEN, Berlin.

Translated from *Arch. f. Augenhlk.*, Vol. LXIX., No. 4, 1911.

(With two figures on Text-Plate XIII. of the German Edition)

AFTER the communication of Wessely, before the meeting of the German Ophthalmological Society, in Heidelberg, in 1910, the question of the possible tuberculous origin of Parinaud's conjunctivitis has aroused fresh interest. Wessely reported on a butcher boy who suffered from a clinically typical Parinaud's conjunctivitis. Implantation of the diseased glandular structure took in monkeys, and the histological and biological examination of these monkeys showed a tuberculous lesion, probably of a bovine type. These observations were confirmed by the clinical experience of Schreiber, Brückner, Wagenmann, and Siegrist.

Several months ago we had the opportunity of observing two cases of Parinaud's conjunctivitis. Before our observations were completed, Stuelp reported on a case of Parinaud's conjunctivitis, and brought strong proof of its tuberculous nature.

Case I.—H. S., 12 years old, of healthy parentage; two brothers are healthy. The boy has had measles—has been otherwise well. Ten days ago the patient suffered from fever, and the right eye became inflamed. The mother noticed a red tumor in the region of the lid, and brought the boy to the hospital. The child has never associated with cattle, though he has played with a cat. The boy is healthy; there are some enlarged glands. The preauricular gland in front of the right ear is enlarged, but not sensitive. Physical examination reveals nothing abnormal, except possibly the percussion note over both apices is somewhat

diminished; breathing is somewhat diminished over this area, on the left side; on the right side expiration is somewhat prolonged.

On examining the right eye there is moderate blepharospasm, and the conjunctiva is injected. On everting the right lower lid, there is a large hypertrophic swelling which is situated between the eyeball and the lid. The swelling is covered by a number of yellowish-white nodules. The eye is otherwise normal. Wassermann test negative; von Pirquet negative. On *Feb. 10th*, 1 milligram (0.001) of old tuberculin was given subcutaneously.

February 12th, evening temperature, 38.2°, no local reaction.

February 13th, normal temperature.

A second subcutaneous injection of the same amount of old tuberculin was given, followed by a rise of temperature up to 38° on the following day, but without any local reaction.

February 18th, no fever. A second von Pirquet test made, strongly positive.

On *February 18th*, one drop of $\frac{1}{100,000}$ old tuberculin is instilled in both conjunctivæ without producing any local reaction during the next day.

On *February 20th*, a drop of $\frac{1}{10,000}$ solution is instilled without causing any local reaction in the next thirty-six hours.

On *February 22d*, a $\frac{1}{1000}$ old tuberculin instillation is made, which also produced no local reaction.

On *February 27th*, a piece of the hypertrophic conjunctiva is excised. A part is implanted into the anterior chamber of a guinea-pig. The rest is sent to Koch's Institute for diagnosis.

On *March 8th*, the patient is discharged cured. Two weeks later the glandular swelling is very much diminished; the conjunctiva is normal.

The implantation in the guinea-pig showed the following: This guinea-pig, after an intracutaneous injection of 0.02 old tuberculin, showed a completely negative intracutaneous reaction.

On *February 27th*, the tissues removed from the patient were put into the anterior chambers of both eyes.

On *March 2d*, there was a corneal opacity and a small prolapse of iris.

On *March 14th*, the animal received another intracutaneous injection of 0.2 old tuberculin. On the following day the site of the intracutaneous injection was strongly positive. It was red, swollen with necrosis on the following days; the controlled animal was negative.

On *April 12th*, the animal died without typical tuberculosis having developed in the eyes. The particles were found surrounded by a fibrous exudate, and there was a plastic iritis. The anatomical examination of the guinea-pig showed no evidences of tuberculosis.

CASE II.— M. S., of healthy parentage, and with no particular previous illness, has had trouble off and on with phlyctenular kerato-conjunctivitis. At present the eye condition has been somewhat worse for two weeks, the glands on the left side have enlarged, and there has been some fever. The child is poorly nourished, rather pale, the cervical glands on both sides are enlarged, and there is a glandular swelling in front of the left ear.

In addition to the evidences of a phlyctenular conjunctivitis, the left eye presents the following condition: The conjunctiva is very red. Between the lower lid and the eyeball there is a hypertrophic swelling about *2cm* broad, situated on the transition fold. On the swelling there are a number of isolated yellowish-white nodules. The Wassermann test is negative, von Pirquet's strongly positive. The treatment consisted in boric acid and silver nitrate.

January 30th 1 milligram of old tuberculin was injected subcutaneously, without producing any rise of temperature or local reaction. This was repeated on February 3d, with negative effect.

On *February 13th*, a third injection of the same amount was given. The temperature rose to 38.5°; there was no local reaction.

February 18th, one drop of old tuberculin, $\frac{1}{100,000}$, was instilled in the conjunctiva, without any effect.

On *February 20th*, a drop of $\frac{1}{10,000}$ solution of old tuberculin was instilled, and there was no local reaction during the next thirty-six hours.

On *February 23d* a $\frac{1}{1000}$ solution of old tuberculin was dropped in the eye, also without any effect.

February 27th, a piece of the hypertrophic tissue was excised, some of it was introduced into the anterior chamber of a rabbit, and the rest was sent to the Koch Institute for diagnosis.

On *April 5th*, the child was discharged cured, except for some opacities from the phlyctenular keratitis. The preauricular glands are still enlarged.

The transplantation was made on *February 27th* to a guinea-pig, which did not react after an intracutaneous tuberculin test, thus proving the animal to be free from tuberculosis. On *March 2d*, the animal died. The particles

of tissue implanted in the eyeballs seemed to be free from reaction. The general autopsy showed nothing.

On *May 12th* the pre-auricular swelling had almost disappeared.

The examination of the tissues, which had been sent to the Institute for Infectious Diseases, showed a negative result in the first case; in other words, no tuberculosis developed, while in the second case, a very weak tuberculosis resulted.

These two cases, with their febrile onset, pre-auricular glandular swelling, one-sidedness, the association with animals, together with the clinical picture of the conjunctiva, unquestionably confirm the diagnosis of Parinaud's conjunctivitis. We did not undertake any histological examination, as we wished to use all material available for inoculation. The many histological examinations which have been made have not revealed any characteristics of a tuberculous structure, though the animal experimentation has been positive. The inoculation experiments have shown in both cases that virulent tubercle bacilli must have been present in the excised tissue, though they were unquestionably very few. In Case I. tubercle bacilli could not be found, though after the implantation of the material into the anterior chamber of the guinea-pig, the previously negative intracutaneous tuberculin reaction became strongly positive. This according to the experimental studies of one of us is very important in the estimation of the infection with virulent tubercle bacilli. Of interest in these two cases are, furthermore, the results of the biologic tuberculin diagnosis, because these are, in a certain respect, opposed to the inoculation symptoms. In both cases the cutaneous reaction of von Pirquet was positive. In both the subcutaneous tuberculin test was positive. This shows in both cases that the body at some time or other had been infected with tuberculosis. This result, of course, does not prove anything as regards the process in the eye, because here alone a local reaction is necessary. In both cases, notwithstanding the general reaction, every vestige of a focal reaction at the site of the conjunctival lesion was absent. The repeated conjunctival instillation of tuberculin in ascending doses also gave no evidences of a local reaction. We must believe that the biologic reaction does not prove the tuberculous nature of the conjunctival lesion.

The inoculation showed the presence of a few tubercle bacilli in the tissue.

The principal result of the experimental tuberculin investigations seem to point to a tendency to form a relative immunity after a tuberculous primary infection against a tuberculous reinfection. The idea of a conjunctival tuberculous reinfection of an organism which had already been infected with tuberculosis is proven by the positive cutaneous and general reactions and the symptoms of the clinical picture, stormy onset, arrest in the first glandular swelling, spontaneous recovery. The cutaneous and subcutaneous tests have shown that a tuberculous infection and a consecutive specific adaptation of the body had taken place. The conjunctival lesion could not be the primary seat of this specific adaptation, because in the presence of a general reaction all local signs were absent. If the conjunctival disease is to be regarded as a tuberculous one, it can only be explained as a local tuberculous reinfection in a body which had previously become tuberculous. This interpretation of a reinfection receives a further proof by the associated phlyctenular process, which we do not regard as an accidental association.

From the present clinical and experimental view on the genesis of the human phlyctenule, it can be taken for granted that it is not itself tuberculous, but it is a local reaction to various specific injuries to the eye in a body which has been previously infected by tuberculosis.

A CASE OF EVERSION OF THE PIGMENT LAYER OF THE IRIS.

By Dr. R. HACK, Würzburg.

Translated from *Arch. f. Augenhlk.*, Vol. LXVIII., 1910.

(With two colored illustrations on Text-Plate X. of the German Edition.)

FRANKE, in 1886, drew attention to the radiating fissures of the pupillary portion of the iris after contusion of the eyeball. This publication was followed by the report of a number of similar observations. They give, in general, a survey on the number and the depth of these tears, their complications in reference to injuries to deeper structures of the eye, and the mechanics of the injury. Though these tears seem to be comparatively frequent, isolated ruptures of the pigment layer of the iris, following injuries of the eye, are unusual.

Gelpke was the first to draw attention to the traumatic coloboma of the pigment epithelium of the iris. He observed in a perforating injury of the eye by a sharp piece of iron, in addition to other injuries, a *2mm* broad fissure which extended in a radiating direction to the ciliary insertion of the iris, that is behind the iris, leaving the pupillary margin intact. Boerma has also described two cases after perforating injury. In one case the fissure extended from the pupillary margin to the root of the iris, its parallel edges were about $\frac{1}{2}mm$ apart. In the second case there were two fissures of varying intensity, in the pigment layer, which could be recognized by illuminating the lens, which was situated posterior to them. By concentration of the light in the focal illumination, no red reflex could be obtained through the pigment coloboma on account of the associated traumatic cataract.

I should like to report on a case which is worthy of interest

on account of its great rarity and for which I have been able to find only one similar condition described in literature

A farmer, 21 years of age, came to our clinic for glasses. On inspection a black thread was found to project free into the anterior chamber out of the pupil of the left eye; and this moved with movements of the eye. The eye was completely free of any irritation, and the tension was normal. In the outer and lower quadrant there was a very small, delicate, horizontal scar, to which the iris was adherent at its pupillary margin. The pupil was consequently distorted and drawn towards this scar. The synechia was discolored on account of the deposit of curious brownish-black pigment. The iris was tremulous in its outer half. The pupil promptly dilated with atropin. From the pupil a long, brownish-black thread hung out of the pupil down to the bottom of the anterior chamber on the anterior surface of the iris. On examining this structure with the magnifying glass, it seemed to be made up of a conglomeration of branching varicose tortuosities, which accompanied every movement of the eye, and seemed to project distinctly from the posterior surface of the iris. Somewhat external from this place a small mass of pigment projected into the pupillary region. The lens presented a few opacities, and in the part near the adhesion there were some vitreous opacities, and the eye was myopic. The right eye was normal, and was also myopic. That this condition could only be the result of a perforating injury, seemed perfectly clear.

The father remembers that fifteen years ago the son was injured in the left eye, and some fluid was said to have escaped from the eye. The eye remained red for some time, and later he noticed this black thread in the eye. This hanging thread in the pupil could only belong to the pigment layer of the iris. The correctness of this was easily demonstrated by the use of the Sachs lamp. The pupil immediately became red, and almost the entire outer and lower quadrant of the iris, though the red light which shone through this quadrant of the iris was somewhat obscured.

The mechanics of this injury can be explained as follows: The blade of straw probably perforated the cornea, and struck the iris near its pupillary margin, perforated this, and reached up to the pigment layer. This layer was thereby separated from its underlying structure, and on account of its unelasticity, the posterior layer of the iris was torn; the pigment strip, being fixed at the pupillary margin, was then everted

through the pupil by the escaping aqueous. The perforating injury then healed, with the production of an anterior synechia.

Gelpke assumes that in his case the traumatic pigment coloboma was the result of an oblique perforation of the cornea by the piece of iron between the pupillary margin and the anterior lens capsule which mechanically removed the pigment layer of the iris.

Boerma believes that the inelastic pigment layer ruptures on account of the pressure exerted by the displaced lens. This pressure the anterior layer of the iris easily follows.

The only analogous case which I have been able to find in literature is a short account by Praun. In a woman the only result of a contusion of the eyeball by a piece of wood was the eversion of the torn-off piece of the pigment layer of the iris through the pupil. This folded flap looked like a carpet hung out of a window.

No mention is made of the transillumination of this part of the iris.

I would like to mention that Vüllers, in 1907, drew attention to the absence of the retinal pigment layer of the iris after inflammatory processes in the uveal tract, and recommends Sachs's lamp for their demonstration, as well as in cases of injury of the posterior layer of the iris.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE NEW YORK ACADEMY OF MEDICINE.

By Dr. MARTIN COHEN, Secretary.

MEETING HELD OCTOBER 21, 1912.

Dr. Rudolf Denig presented a patient with an **ammonia burn of the eye.** As in the two other cases of lime-burn presented to the Section in the spring of 1912, he performed a circular excision of the burned conjunctiva around the limbus on the fifth day after the accident, and covered the defect by a large circular lip flap. Within three weeks after the operation the cornea cleared up nearly entirely.

Dr. H. H. Tyson inquired whether the cornea was involved, to which Dr. Denig replied that it was not. Dr. Tyson then related a case of severe burn of the conjunctivæ, corneæ, pharynx, tissues of the eyelids and face. It occurred when a patient in epileptic coma unexpectedly moved his hands, dashing the ammonia held before him to inhale, into his eyes and nares, so that it trickled into his pharynx. On the second day after the accident, when called in for consultation, he found a dense keratitis striata and iritis, and both corneæ very hazy. The eyes had been previously bathed with boric acid solution. Vision O. D. and O. S. was barely movements of hands. Atropin in olive oil was instilled and a 1 per cent. boric acid ointment used freely at first, weak iodoform ointment later. After about six weeks the eyes were healed and vision was O. D. $\frac{20}{40}$, O. S. $\frac{20}{40}$ with correction.

Dr. Talbot R. Chambers reported a case of **regeneration of corneæ lost in ophthalmia neonatorum.** A boy, one day old, had sore eyes. The Credé method had been employed.

Gonococci were found in child and both parents. The child appeared healthy and strong otherwise.

Argyrol instilled and the eyes were left undisturbed for fifteen minutes. A boric solution was allowed to flow between the lids, night and day. Ice compresses. In spite of atropin, pupils became pin-point. Four days later: Ice and atropin discontinued. Six days later: Anterior wall of both eyes almost melted away. No anterior chamber. Four days later: Right iris prolapsed. Next day: Corneæ growing from the periphery. Four days later: Right cornea complete and transparent, but left had a large central macula. Anterior chambers about half normal depth. Child died later from effect of hot weather.

Dr. Chambers also presented this statement in substantiation:

"This certifies that the Demato child when one day old, on May 29, 1912, became a patient in Christ Hospital, Jersey City.

"That on June 8th or thereabout, the cornea of each eye had melted away and there was no anterior chamber and the iris was the anterior wall of each eye. There was no pupil or rather it was pin-point in each eye: that on June 12th, the cornea began to grow from the periphery in each eye.

"ANTHONY G. SACCO, M.D.
"HAROLD W. BROWN, M.D.
"M. E. BAXTER, M.D."

The child was seen by the doctors in an Italian Dispensary in New York City and they recommended some operation to restore sight—about June 15th.

Dr. JULIUS WOLFF had had a similar case with an infected corneal ulcer, causing a prolapse of the iris of about ¼ inch. Conjunctiva chemotic. After application of wet dressings and compression, the mass became gradually smaller, finally resulting in thin, transparent, corneal tissue.

Dr. J. H. CLAIBORNE remarked that Dr. Peter Callan had reported a similar case several years ago.

Dr. C. W. CUTLER thought that the cornea could not very well become transparent after such complete destruction of the corneal tissue.

Dr. OTTO SCHIRMER said the tissue could not become trans-

parent in regeneration of the cornea, as Bowman's membrane was never restored, and that the scar tissue would cause permanent opacity in later years.

Dr. W. B. WEIDLER reported a case of **large papilloma of the cornea** with microscopical demonstration of growth. Patient was a fisherman, aged 61. Family history negative as to malignant growths; personal history negative. Had noticed three years ago a redness over the left eyeball at the interpalpebral space, which six months later had assumed the present dimensions: 12*mm* long, 8 wide, and 8 high. Freely movable, dense and firm to touch. Uneven and very vascular surface. The epithelial layer of the cornea seemed to be invaded. The growth was neither painful, nor tender, did not bleed freely and appeared to be limited to the cornea. The tumor was said to have grown rapidly. No glandular enlargements about ear or neck. Vision O. D. $\frac{5}{5}$, O. S. $\frac{5}{5}$. A month later the eye was reported to be getting white and to give no trouble whatever.

Microscopical Examination.—The outer two-thirds of the tumor showed a very large papilla-form arrangement. It is invaded throughout by a formation of new blood-vessels which do not show the typical walls. Several hemorrhagic areas are present in sections. Collections of very small round cells are seen in different sections, one near the center being larger than the others. Between the cells there are some septa of connective tissue. There are neither giant cells nor cell nests. This portion of the growth suggests the possibility of a carcinomatous change taking place in or near the center. The epithelial layer of cells in the small dissected strip are much hypertrophied and thickened by connective tissue stroma of low development.

Dr. EDWARD A. DAVIS had a similar case, in which fibroma was demonstrated by microscopical examination. There had been no recurrence after several years' observation.

Dr. WALTER E. LAMBERT presented a patient who had a large and ragged **foreign body in the vitreous** removed by a scleral incision and the aid of a small magnet. The foreign body was located by Dr. Dixon in the lower and outer quadrant. The vitreous was cloudy. Lens clear. There was a subconjunctival hemorrhage. Recognizing the danger of

detachment of retina as pointed out by Prof. Haab, he considered it might be disastrous to the eye to remove the foreign body by the usual method, being so very large and ragged. Vision to-day, after a month, is $\frac{20}{70}$ without any retinal detachment; and the result up to-date appears to be excellent, although the future will tell its effect.

Dr. OTTO SCHIRMER said he preferred removing foreign bodies in vitreous through the anterior chamber. If large, he used at first mild power, and then, if necessary, applied the magnet for varying periods of time for several days.

Dr. CLAIBORNE said the interest in the case, although excellent at present, would lie in the future.

Dr. W. B. MARPLE presented a patient with **retro-orbital tumor,** whose sight had been gradually impaired for the past two years, exophthalmos appearing recently. Counted fingers at two feet. Ophthalmoscopic examination indicated optic neuritis. There was no ocular palsy. The tumor is probably of slow growth.

Dr. CLAIBORNE asked if there was any accessory sinus involved. Dr. Krug said that a rhinological examination would be performed shortly.

Dr. EAGLETON said he had had a patient with a mucocele of the ethmoid which had given no pain; there was distinct exophthalmos.

Dr. H. H. TYSON presented a case of **ptosis following a Heisrath operation for trachoma**. A. B., 21, applied at the Vanderbilt Clinic for the relief of ptosis. She had been previously operated upon for trachoma, a portion of the conjunctiva having been excised. Immediately after the operation the upper lid drooped, a condition which had never existed before.

Several such cases had been noticed in Germany, but none had been presented at the Section. The present case did not detract from the merits of the operation itself, but showed a possible result of it when performed with imperfect technique.

Dr. CLAIBORNE thought that the section of the retrotarsal fold in this case was too far back, and that he had not encountered such a result in any similar operation performed either by himself or others.

Dr. EAGLETON reported a case of **congenital tumor of sclera and cornea.**

Dr. W. B. WEIDLER thought it looked like a lipoma and advised microscopical examination of a piece of the tumor.

Dr. W. B. WEIDLER presented a 3-year-old, well nourished healthy girl with a **double optic atrophy,** primary, probably due to syphilis. Mother had one miscarriage and died of tuberculosis. Patient had measles and whooping cough. Mind bright and clear. Eighteen months ago she fell on her head, and there is an indefinite story of convulsions. About a year later vision failed and has remained so.

Pupils: 5*mm*. No reaction to light, accommodation, or divergence. Media clear. Scleral ring is pale white with some atrophy of choroid. Vessels are small and contracted. General atrophic changes in fundus. Slight lateral nystagmus. Urine negative, Wassermann positive, knee jerks absent, station and gait normal. Von Pirquet negative. X-ray of skull failed to show any abnormality. The history of miscarriage, positive Wassermann, absence of knee jerks and of secondary symptoms in the fundus, as seen after meningitis, made the above diagnosis tenable.

Dr. W. M. LESZYNSKY considered the history faulty and thought that the spinal fluid should be examined to rule out syphilis. The condition might be due to pressure in the inter-peduncular space.

Dr. W. B. MARPLE presented a set of Dr. Ballantyne's **aseptic dropping tubes.**

Dr. TALBOT R. CHAMBERS described a new **lamp for retinoscopy.**

Light, which is satisfactory for ear, nose, and throat is too bright for eye work. The author has devised a three-armed bracket to suit all cases without the escape of light except through the intended opening. It consists of a 40 Watt electric bulb, a brass cylinder to hold it, with an opening outside of which is a raised groove for insertion of a smoked lens. This will allow the pupil to dilate. With the lens removed, the observed pupil will at once contract.

Dr. MARK J. SCHOENBERG read a paper on **Clinical and experimental researches on intraocular pressure**. (Published in full in this issue.)

Dr. TYSON, having known the purport of Dr. Schoenberg's paper for some two weeks, had made a few tests, from the result of which he could confirm Dr. Schoenberg's findings. He hoped the author would be able to formulate a uniform rule as to the rate of decrease in tension with more extended observation and investigation, which would be of great value as an aid in diagnosis in doubtful and prodromal glaucomas. The rate of return of increased tension was also an important point to establish, and he thought Dr. Schoenberg would find a similar disparity. The facts brought out in this paper were worthy of investigation.

Dr. SCHIRMER hoped the work, although new, would at some future date be utilized to good advantage, and thought it would still require more work to prove certain principles.

Dr. CUTLER asked what anæsthetic was used; whether the tonometer could be used on closed eyes; whether the cornea was damaged, and whether the application of the tonometer did not inhibit the secretion of the intraocular fluid, thereby affecting the tension.

Dr. SCHOENBERG, in closing the discussion, stated that the anæsthetic used was holocain; that the application of the tonometer did not damage the cornea; that using the instrument on closed lids was not accurate, as this affected the tension of the eyeball. As to the instrument inhibiting the intraocular fluid, he could not answer at present.

MEETING HELD NOVEMBER 18, 1912.

Dr. H. H. TYSON presented a case of **lipodermoid** in a negro girl, aged 15. The tumor was noticed at birth and had gradually increased in size. It was yellowish in color and was located at the outer circumference of the left eyeball, overlying the external rectus. It consisted of two large lobules, the larger being below, and both extending deep into the orbit behind the outer canthus. The conjunctiva covering it appeared greatly thickened like skin, and had a greasy reflex with several hairs growing upon it. On account of the irritation produced by the hair, and for cosmetic reasons, it was advised that the tumor be removed.

Dr. GRUENING said that these tumors do not increase in size.

Dr. TYSON replied that he had only seen the case recently,

and he accepted the statements of the mother. These agree with the experience of Fuchs, that these tumors sometimes increase considerably in size around puberty, and he thought that in this case the mother's observation and statement were correct.

Dr. WEEKS presented two patients with the condition known as **family nodular keratitis;** one the father, a man of 56 years, the other a son of 23 years. The peculiarity of each was a punctate or short-lined, elevated opacification on the cornea, gradually lessening in density toward the periphery, terminating about *2mm* from the margin of the cornea. The condition was the same in both patients except that it was more pronounced in the elder. The vision of the father was $\frac{20}{70}$, of the son $\frac{20}{40}$. The affection had been noticed in early youth in both, and had very slowly advanced. There had been no inflammation and no pain. The father had had three brothers and five sisters. He reports that two of the sisters and one brother had poor vision, but he did not know the nature of the trouble. Besides the son here presented, there were two others; one, aged 18 years, had been examined and the eyes found to be normal, the other son was reported to have poor vision, but as he was in another city the nature of the trouble could not be determined. Dr. Weeks spoke of the appearance and pathology of this affection as follows: The opacification begins from the twelfth to the eighteenth year and slowly increases in density. Males and females are affected in about the same proportion. The opacity is greatest at the center of the cornea, never reaching the limbus. The development is extremely slow, beginning as minute dots or lines. "Examination at an early stage shows that the opacity is made up of little light gray streaks and dots, which by transmitted light appear transparent like threads of glass and little drops of water on a glass plate." Fehr is of the opinion that the changes are due to the development of hyaline masses in the superficial lamellæ of the cornea, in which the crystals of phosphate of sodium urate are subsequently deposited.

Dr. OATMAN said that these cases belong to a class which appears in certain families. It is most frequently called "gitterige keratitis" from a tendency of the lesion to form

superficial grill-like opacities on the cornea. It also assumes the form of lines, dots, etc., and then is named according to the shape of the opacities.

Dr. H. W. WOOTTON presented a patient, male, 45, with **bilateral proptosis** which was first noticed about eight months ago. It was now very marked, the orbital fat being dislocated and readily visible on slightly elevating the lids. The latter can, however, be closed. No history of injury, no goitre, no tachycardia, no psychic symptoms. There are fibrillar tremor and motor limitation of the globes. Fundi normal. Vision for form normal, for colors contracted. Accessory sinuses exceedingly large, but apparently clear. Wassermann negative. Treatment with urotopin, resulting in some improvement. The diagnosis rests upon the proptosis and tremor, together with the fact that no other cause can be assigned for the exophthalmus.

Dr. JOHN GUTTMAN presented a case of **polyp of the conjunctiva.**

Dr. KELLNER presented and demonstrated the use of the following instruments: (1) Large ophthalmoscope after Gullstrand; (2) Hertel's exophthalmometer; (3) binocular magnifier; (4) monocular and binocular telemagnifier.

Dr. ARNOLD KNAPP read a paper entitled **Retinal changes in adolescence,** which appeared in full in last issue of these ARCHIVES.

Dr. OATMAN stated that, according to Michel, recurring intravitreous hemorrhage of young adults is often the first symptom of tuberculosis of the uvea. Axenfeld asserts that there may be tuberculous disease of the intraocular vessels without visible fundus lesions. He had seen a case of recurring intravitreous bleeding which recovered under tuberculin injections.

Dr. WEEKS said that the opinion that retinal hemorrhage and the subsequent development of some cases of retinitis proliferans in adolescence was due to tuberculosis was relatively new and was gaining ground. The opinion that bacterial infection of other forms was responsible for retinal hemorrhage in some cases and that local endarteritis was responsible in some others was believed by some observers. That the development of connective-tissue bands in the vitreous must

be preceded by the occurrence of a clot in the vitreous with or without the presence of red blood corpuscles is conceded by nearly all observers. It is also known that not all the formations of clots in the vitreous are followed by the formation of connective tissue bands. It would, therefore, follow that a condition favorable to the development of new connective tissue must exist. What determines this favorable condition is not known, but that the development of connective tissue bands constituting the condition known as "retinitis proliferans" may follow "idiopathic" hemorrhage, hemorrhage due to traumatism, and hemorrhage in syphilitic retinitis and chorio-retinitis is well known. In cases of "idiopathic" retinal hemorrhage and retinitis proliferans the possible tuberculous nature should be kept in mind and, if this can be substantiated, treatment with tuberculin should be instituted.

Dr. MARPLE has had a similar case with vitreous hemorrhage in a young person. While the treatment and prognosis were unsatisfactory, still he thought a suggestion was given in this paper as to its origin.

Dr. REESE recalled a case with a tumor-like mass of the choroid which gave a positive general tuberculin reaction; however, the therapeutic tuberculin treatment arrested the process.

Dr. ROBERT G. REESE presented a paper on **Dystrophia epithelialis corneæ,** stating that the majority of cases are due to local disturbances of nutrition, many of which have, prior to the appearance of Fuchs's monograph in 1910, been erroneously diagnosed as glaucoma. It is a bilateral affection of advanced age, chiefly affecting the female sex; exceptionally, the right eye alone is attacked. The onset is insidious without any manifestations of irritation, both corneæ becoming insensitive. There are variations in vision unattended by variations in pressure, vision being worse in the morning, contrary to glaucoma. According to Fuchs, the changes are characterized by a grayish opacity, roughening of the corneal surface caused by small vacuoles in the epithelial cells, and newly formed tissue between Bowman's membrane and the epithelium, consisting of homogeneous connective tissue without any signs of hyaline or mucous degeneration. Larger vesicles are due to the separation of the epithelium

from Bowman's membrane which occurs later in the disease. The anæsthesia of the cornea is due to the injury of the superficial nerve fibers of the epithelium, the subepithelial nerve plexus, or the fibers connecting the two. The explanation of the pathogenesis is therefore probably a disturbance of nutrition affecting first the nerve and then the cornea, which is supported by the fact that paralysis of the trigeminus is followed by keratitis neuroparalytica.

Glaucoma, which complicates a number of cases, may result from the dystrophia, or there may be an etiological factor common to both. The etiological factor, known so far, is advanced age. Occupation plays no part, and nearly all the patients were in good health and showed no signs of malnutrition.

The affection should be differentiated from opacities caused by noxious vapors. In these cases, however, the lesion is confined to the palpebral aperture and disappears when the noxious agent is withdrawn. Acute keratitis vasculosa occurs in neurotic subjects or together with herpes; in the chronic form it usually complicates some grave eye lesion. Postoperative sclerosis of the cornea in the aged resembles dystrophia, but is probably due to traumatism of the endothelium.

Dr. KNAPP hoped that Dr. Reese would have something new to suggest in the line of treatment in this condition. In his experience, the cases of dystrophy had occurred after a cataract operation in senile patients. He thought at one time that a section entirely in the cornea might have something to do in bringing on this condition; but in one of the two cases observed the section was a scleral one with a conjunctival flap. The process in these cases seemed to be a lack of regenerative power of the corneal nerves, which had been divided at time of operation. There is one important feature in this connection, and that is, if one eye develops this condition after a cataract operation, no operation should be done on the other eye.

Dr. WEEKS. The term dystrophy, meaning changed or ill nutrition, may manifest itself in many ways. In the epithelium of the cornea it may be an atrophic change due to senility, to disease of the Gasserian ganglion, or due to trau-

matism. When due to senility it may appear in the form of ribbon or band-shaped keratitis, or it may affect the entire cornea. When due to disease of the Gasserian ganglion, it will affect the cornea corresponding with the ganglion affected and will usually involve the entire cornea. When due to traumatism, it usually affects the entire cornea. In all cases the corneal surface is roughened. The cornea may exfoliate. There is loss of sensibility. Dr. Weeks had observed a number of cases. Treatment did not appear to be of much avail.

Dr. W. B. MARPLE gave a brief report of the Oxford Ophthalmological Congress of this year. As to Professor Straub's (Amsterdam) paper on the doctrine of scrofulosis, he did not think that the lecturer had presented any very good reasons for continuing the word "scrofulosis." The discussion on coal miners' nystagmus brought out the almost unanimous opinion that this was due to the defective illumination from the Davy safety lamp. In mines where unprotected lamps were permitted, nystagmus was almost unknown. Van Lint's (Brussels) sliding flap operation in the removal of cataract claims to diminish the liability to infection and prolapse. But as this is a rare complication to-day, Dr. Marple did not feel inclined to use it.

A pleasant feature of the program was that there were only a few papers, with an interval between them, which gave the hearers an opportunity to look over the exhibits and to investigate new preparations and devices. Among the exhibits there were a collection of beautiful colored drawings of 250 mammalian and reptilian eyes, by A. W. Head, Maddox's useful device for locating the axis in patients' glasses, and Thomson's beautiful macroscopic preparations of the eye.

Dr. Marple concluded by stating that Oxford was at present a splendid place to study anatomy under Professor Thomson and his assistants.

Dr. KNAPP thought that the great advantage of the Oxford meetings was that the meetings were held in university buildings with laboratories, so that every facility was given for the proper demonstration of specimens, methods of examination, and apparatus. The lack of this is especially felt in the summer hotels, where most of our meetings are held.

REPORT OF THE PROCEEDINGS OF THE SECTION ON OPHTHALMOLOGY OF THE ROYAL SOCIETY OF MEDICINE.

By Mr. C. DEVEREUX MARSHALL, London.

THE PHYSIOLOGY OF THE INTRAOCULAR PRESSURE.

At a meeting of this Section on Wednesday, December 4th, under the Presidency of Sir Anderson Critchett, C.V.O., the subject for discussion was **The physiology of the intraocular pressure.**

Dr. Leonard Hill, F.R.S., in opening the debate, said the reason Dr. Flack and he appeared before the Society was because some researches which they had been carrying out on the relation of the general circulation to secretion led them to investigate the eye. The subject had already been brought before the Royal Society, but there were some new points which he would speak on. For the purpose of measuring the intraocular pressure, Mr. Thomson Henderson and he made some experiments because with the ordinary hypodermic needle they could not avoid leakage in the track of the needle. The result was, they devised a needle which did not permit of leakage. They found, by very carefully checked experiments, that the aqueous pressure varied within fairly wide limits, depending on the blood pressure: the higher the arterial pressure, the higher was the aqueous pressure. In one case, under chloroform, it was 16*mm* of mercury, and under ether narcosis, when the animal was in good condition, going up as high as 62*mm*. By a compensation method they obtained readings which seemed to agree, so there seemed but little chance of error. It was very important that the pressure of the aqueous should be positive, because that kept the eyeball as a perfect optical instrument. Its distension, in their

opinion, was maintained by secretory pressure. Though the pressure varied anywhere between 10 and 50*mm* according to the condition of the circulation, the eyeball retained its shape. The pressure in the capillaries and the pressure of the aqueous must always be the same. In a previous investigation they found that the pressure of the cerebro-spinal fluid and the pressure of blood in the cerebral veins were the same, and the two varied together. Various workers had made investigations and it was found by viewing the retinal vessels with the ophthalmoscope that the flow ceased in them when the pressure reached that of the general arterial blood. Dr. Hill and his colleague punctured the cornea of a cat, and then on gently pressing the abdomen the iris on the same side immediately burst into hemorrhage: all the capillaries allowed the blood to escape, and that came off every time. The explanation was that the normal aqueous pressure was exactly counterbalancing the capillary pressure. The aqueous had a definite chemical nature, and its osmotic pressure was said to be higher than that of the serum. Ehrlich demonstrated, by the injection of fluorescene, that some kind of circulation was proceeding in the aqueous. It seemed, from careful inquiry, that the cribriform ligament was opened up, and thus a passage was made for the fluid into the supra-choroidal space. That transference of fluid would allow accommodation to take place. The suspensory ligament was always taut, exerting elastic traction on the lens and causing it to assume its proper shape. The intraocular tension was said to be due to the elasticity of the lens, but it had no elasticity, for when removed it was a soft, pudding-like body. In the act of accommodation the muscle contracted, but continued to be of the same volume as the uncontracted muscle; on contraction it really moved inwards, as described by Thomson Henderson, and allowed the fluid to pass from in front of the lens either into the spaces of the ciliary body, or into the space of the cribriform ligament and into the supra-choroidal space.

Professor Starling, F.R.S., said most would agree with the facts which Dr. Leonard Hill had advanced, but he was not in close agreement with some of the interpretations which the opener placed upon them. It was important to have some idea of the factors determining the formation and

absorption of the intraocular fluid, but more important to know what those factors were. It seemed to be generally agreed that the seat of production of the intraocular fluid was the ciliary processes, and the chief absorption was at the anterior angle of the eye, and some probably occurred at the root of the iris.

Covering the ciliary processes was a well-marked epithelium, containing columnar or cubical cells, which might well be endowed with the property of secretion, as they were as well formed as those of the salivary gland, or of the kidney tubules. What were the conditions which must be observed if the intraocular fluid was to be regarded as a filtration? The intraocular fluid was at considerable pressure, so there must be resistance against its flow from the eyeball. But if the pressure there was in consequence of the pressure of the blood in the vessels, the pressure of the intraocular fluid must go up with the rise of pressure in the vessels. When the carotid or subclavian artery was obstructed, cutting off the blood supply from the eyeball, the pressure came down; and it went up again in proportion to the amount of blood one allowed to enter those vessels. One could take 25*mm* as the average intraocular pressure. Stimulation of the sympathetic nerve caused a double effect: first, contraction of the unstriped muscle fibers of the orbit and a rise of pressure, and as the vessels of the ciliary process contracted so as to diminish the blood pressure in those vessels, there was a fall also in the intraocular pressure. There was a quick rise due to contraction of the nictitating membrane and the unstriped muscle fibers, and a rise of pressure. As the formation of intraocular fluid passed off, there was a fall of pressure, accompanied by constriction of the vessels of the eyeball. He contended that the blood pressure must be higher in the vessels all the way along than outside and than the intraocular pressure, and the quicker the rate of flow through the system of vessels concerned, the greater must that difference of pressure be. There was no reason for saying it was impossible for a difference of pressure to exist between the capillaries and the fluid outside; but there was every reason for assuming there must be a difference. One could not know how much fluid came out until one ascertained what was the rate of transudation. If the pressure

remained constant, that rate was the rate of absorption. Professor Starling showed a number of slides to illustrate his point. It was impossible to measure the pressure in the capillaries, under normal conditions. By increasing the pressure of the intraocular fluid one brought up the pressure in the capillaries towards that in the arteries. At some future time it might conceivably be shown that the cells coming from the ciliary processes might act as regulators, but at present there was no evidence that they acted otherwise than as guiding and strengthening the filtering membrane. In regard to the relationship of the capillary to the venous pressure, he had never been able to understand Dr. Leonard Hill's position on that point. Into the mechanism of accommodation he did not propose now to enter, except to say he supposed it to be due to a shifting of fluid between the posterior chamber and the anterior chamber, which could easily take place, as there was no resistance between the fibers and the suspensory ligament.

Mr. PRIESTLEY SMITH (Birmingham) said it was important to know what was the normal pressure in the human eye. It certainly varied in different people and in the same person at different times; but what would be a fair average? A pressure of 60*mm*, which had been mentioned, would surely mean that the person had glaucoma. Dr. Hill had said that the intraocular pressure was equal to the venous pressure, and to the pressure in the capillaries. He, Mr. Smith, thought it was equal to the venous pressure at one point, *i. e.*, where the blood left the eye, but that at every other point the intraocular pressure was lower than the blood pressure. Dr. Hill had not shown that the pressure in the torcular herophili was equal to the pressure in the veins and the other parts of the skull; and, as Professor Starling said, there could not be equality of pressure or there would be no movement. But the difference in pressure was probably only very slight. He did not see how Dr. Hill could hold it as proved that the pressure in the capillaries of the ciliary body was the same as the pressure of the intraocular fluid; and every probability seemed to be against that view. It was generally acknowledged that the intraocular pressure was produced by the ciliary processes and he believed the ciliary body also played a part.

There was no question that the vitreous fluid was renewed, but did it come from the posterior surface of the process, or from the *pars plana?* He expressed his objection to the use by some, even chemists, of the term "tension" instead of "pressure." There had been a controversy as to whether the tension of the eye depended on increased volume, or on increased pressure of the blood. Such a discussion was futile, because it depended on both.

(The discussion was postponed until the next meeting of the Section.)

QUARTERLY REVIEW OF THE PROGRESS OF OPHTHALMOLOGY.

By H. KÖLLNER, Berlin; W. KRAUSS, Marburg; R. KÜMMELL, Erlangen; W. LOEHLEIN, Greifswald; H. MEYER, Brandenburg; W. NICOLAI, Berlin; H. PAGENSTECHER, Strassburg; K. WESSELY, Würzburg; and M. WOLFRUM, Leipsic, with the Assistance of Drs. ALLING, New Haven; CALDERARO, Rome; CAUSÉ, Mayence; DANIS, Brussels; GILBERT, Munich; GROENHOLM, Helsingfors; v. POPPEN, St. Petersburg; TREUTLER, Dresden; and VISSER, Amsterdam.

SECOND QUARTER, 1912.

Edited by Dr. MATTHIAS LANCKTON FOSTER, New Rochelle.

I.—GENERAL OPHTHALMOLOGICAL LITERATURE. Reviewed by WESSELY.

(Books, Monographs, Historical Essays.)

136. LOHMANN. **Functional disturbances of vision.** Leipsic, 1912.
137. WAGENMANN. **Injuries of the eye.** *Graefe-Saemisch's Handbook of Ophthalmology*, parts 219 and 220.

LOHMANN (136, **Functional disturbances of vision**) deals with physiological optics chiefly from the point of view of the changes that appear as pathological symptoms. The main part of the work is contained in the chapter on the pathology of entoptics, disturbances of the light sense, adaptation, color sense, and binocular vision. The section on color hearing should be specially mentioned. The book is a very good guide to the literature on the subject, which is freely quoted.

In these parts of Graefe-Saemisch's Handbook WAGENMANN (137, **Injuries of the eye**) deals with burns, both by high temperatures and by alkalies and acids, with special attention to those produced by lime. Then he passes to injuries by other chemical agents, vegetable and animal poisons, and finally electricity.

KÖLLNER.

II.—RELATIONS OF OPHTHALMIC TO GENERAL DISEASES, INCLUDING POISONS. Reviewed by WESSELY.

138. ANTONELLI. **Polyneuritis of the cranial nerves after treatment with arsenobenzol.** *Société française d'ophtalmol.*, May, 1912.

139. BITTORF. **Disturbances of the internal secretions.** *Berl. klin. Wochenschrift*, No. 28, p. 1072.

140. COPPEZ, H. **Ocular complications of Paget's disease.** *Journal médical de Bruxelles*, June 13, 1912.

141. COUTELA. **Ocular accidents attributed to arsenobenzol.** *Archives d'ophtalmologie*, xxxi., p. 1.

142. ELSCHNIG. **Indicanuria and diseases of the eye.** *Wien. klin. Wochenschrift*, No. 19.

143. ELSCHNIG. **Conclusion of the discussion concerning indicanuria and diseases of the eye.** *Ibid.*, No. 24.

144. FEHR. **The effect of salvarsan on the eye.** *Centralbl. f. pract. Augenheilkunde*, June.

145. FEHR. **The outbreak of syphilis in the eye after treatment with salvarsan.** *Med. Klinik*, No. 23, p. 942.

146. FOERSTER. **Differential diagnosis and treatment of methyl-alcohol poisoning.** *Muench. med. Wochenschrift*, No. 16, p. 862.

147. FUCHS, E. **Tabes and the eye.** *Wien. klin. Wochensch.*, No. 14.

148. GUTMANN. **Diseases of the contents of the orbits after extraction of teeth.** *Berl. ophth. Gesellschaft*, Feb. 29, 1912.

149. LEBER, A. **Tropical diseases of the eye.** *Meeting of the Ophthal. Society of Heidelberg.*

150. MOISSONIER. **Optic neuritis of gouty origin.** *Société française d'ophtalmologie*, May, 1912.

151. PICK, L., and BIELSCHOWSKY. **Histological conditions in the human eye and central nervous system in acute, fatal poisoning with methyl alcohol.** *Berl. klin. Wochenschrift*, No. 19, p. 888.

152. REDSLOB. **Schools and eyes.** *Strassburger med. Zeitung*, No. 3.

153. REICHARDT. **Studies of the brain.** Part 2, Jena, 1912.

154. SATTLER, C. H. **Pathological examination of a case of blindness after injections of arsacetin.** *Arch. f. Ophth.*, 81, 3, p. 546.

155. TERSON. **Ocular lesions in polymorphous erythema; erythema nodosum and tuberculosis; erythema nodosum and glaucoma.** *Arch. d'ophtalmologie*, xxxii., p. 274.

156. TERSON, A. **The dental origin of certain affections of the eye.** *Journal médical de Bruxelles*, April 4, 1912.

157. WIEGMANN. **A case of disease of the optic nerve after whooping cough.** *Klin. Monatsbl. f. Augenheilkunde*, April, p. 460.

According to A. LEBER (149, **Tropical diseases of the eye**) the study of the as yet little known diseases of the eye peculiar to the tropics presupposes a knowledge of the general climatological factors which govern the biological relations of the organism in those countries. To these must be added

the racial biological conditions, which render difficult the nosological comparison of tropical and non-tropical diseases. Exudative inflammations of infectious or toxic origin stand in the foreground among the diseases of the lids. Bacterial are less common than mycotic infections. Framboesia, leprosy, and gangosa cause extensive changes in the lids. In diseases of the conjunctiva the power of regeneration is striking. Forms of conjunctivitis induced by fungi and allied organisms render the clinical pictures to be observed very numerous. Inflammatory intraocular symptoms are met with in dengue, hemorrhages in malaria and anchylostomiasis, and serious degenerations of the choroid caused by emboli in filariasis. Nervous disturbances of central origin have been met with in beriberi and an endemic encephalitis has been observed in Samoa. The stamping out of epidemics of tropical diseases of the eye must be begun by the adoption of hygienic measures.

KR.

REDSLOB (152, **Schools and eyes**) examined in three years about 4000 eyes of school children and found diseases of the lids and conjunctiva in 10% of the cases, of the cornea in 13%, of the lens, retina, optic nerve, uvea, and vitreous together in only about 3%, nystagmus in 1%, strabismus in 7%, and errors of refraction in 68%. The last were hypermetropia in 17%, myopia in 14%, and astigmatism in 37%. The great importance of astigmatism is brought out strongly. Mixed astigmatism was present in only 2% of all cases of astigmatism. He then deals with the prescription of glasses, the surveillance over the children, their choice of occupations, instruction in writing, and the formation of special classes for children with poor eyes which has been undertaken in Strassburg.

GUTMANN (148, **Diseases of the contents of the orbits after extraction of teeth**) has seen three cases of serious orbital trouble following extraction of teeth by dentists who apparently were not sufficiently aseptic. In one case, a child, aseptic thrombosis and bilateral cellulitis of the orbit were caused; in another cellulitis of one orbit with sinus thrombosis; in the third an empyema of the antrum of Highmore which led to a neuritic atrophy of the optic nerve.

According to TERSON (156, **The dental origin of certain affections of the eye**), diseases of the teeth play an important part in the etiology of diseases of the eye. Blepharospasm, abscess of the lid, pareses of the muscles, and hypersecretion of the lachrymal gland are induced in this way; more rarely, inflammation of the iris and ulcers of the cornea. Optic neuritis with or without sinusitis may result from a periodontitis. Inflammation of the maxillary sinus may cause detachment of the retina and retinal hemorrhages; extraction of teeth may give rise to infectious metastases in the choroid. Visual disturbances, varying in degree from a weakness of the accommodation to bilateral amaurosis, may originate in affections of the teeth, and even glaucoma may be excited or made worse by them. Etiological connection is also shown to exist between infections in the orbit, pareses and contractures of the muscles of the eye, and extractions of teeth or inflammations in the vicinity of the teeth.

DANIS.

BITTORF (139, **Disturbances of internal secretions**) reports first a case which presented the symptoms of acromegaly, as well as a dilatation of the sella turcica in the X-ray picture, but who had instead of a bitemporal, a binasal hemianopsia with pale papillæ. As Wassermann's reaction was positive, and as improvement followed the administration of potassic iodide, the trouble is supposed to have been a syphilitic process at the base of the brain in the neighborhood of the hypophysis, which involved the non-decussating fibers of the optic nerve. He also reports a case of dystrophia adiposo-genitalis in which the X-ray picture showed a great enlargement of the sella turcica and a choked disk was found on ophthalmoscopic examination.

WIEGMANN (157, **Disease of the optic nerve after whooping cough**) found in a woman 40 years old, who had had an attack of whooping cough and complained of obscuration of vision, a haziness of the margins of the papilla and venous engorgement. He ascribes the condition found to the long duration of the whooping cough with its often repeated conditions of engorgement of the vessels of the head.

COPPEZ (140, **Ocular complications of Paget's disease**) has found in four cases of Paget's disease little yellowish

patches of degeneration in and about the macula, which caused metamorphopsia and little scotomata. They develop slowly and sometimes are accompanied by punctate hemorrhages. These places blend and finally form an atrophic spot. The optic nerve remained healthy. An incipient cataract was present in each case.

DANIS.

MOISSONIER (150, **Optic neuritis of gouty origin**) observed a typical retrobulbar neuritis in a gouty person 43 years old. Such cases are rarely met with. The visual disturbance consisted of the characteristic pains behind the eye and a central scotoma, while the external and internal condition of the eye appeared to be normal. A cure was attained within twenty-five days by treatment with aspirin and colchicum, bleeding, and subconjunctival injections. Moissonier is inclined to think that retrobulbar neuritis is more commonly of gouty origin than has hitherto been supposed.

CAUSÉ.

The papular, macular, and nodular forms of polymorphous erythema frequently cause diseases of the eye. TERSON (155, **Ocular lesions in polymorphous erythema**) reviews these with special reference to their connection with tuberculosis and with glaucoma. He reports two cases of erythema affecting the eye in tuberculous patients, one macular, the other nodular. He thinks that the nodular form occurs the more frequently with tuberculosis. Glaucoma is excited in erythema by a hypersecretion. The local treatment of the ocular symptoms is usually unsatisfactory; the best results are obtained by dionin. The main treatment must be directed toward the etiological disease.

CAUSÉ.

FUCHS (147, **Tabes and the eye**) says that the assumption that reflex immobility of the pupils is characteristic of tabes, and that absolute immobility is on the contrary diagnostic of cerebral syphilis, is not absolutely correct, because in reality both sorts of pupillary disturbance are met with in both diseases. Tabetic ptosis not rarely exhibits the peculiarity that it increases in abduction of the eye and decreases in adduction, to such a degree indeed that the lid may rise higher than that of the other eye, a phenomenon that is to

be ascribed to an abnormal transmission of the stimulation from the nucleus of the internus to that of the levator in the diseased nuclear region of the oculomotorius. The division of tabetic optic atrophy into two types, one with a uniform loss of central and peripheral vision, the other with sector shaped defects in the visual field with good central vision, is not justified, for the two forms blend with each other. There are only three special types: 1, a minimal central visual field with good vision, exceptionally with preserved color sense, as in glaucoma; 2, a papillomacular scotoma, as in toxic amblyopia, a form observed in 30 of his private cases of tabes, which always led to total blindness in spite of all antisyphilitic treatment, so that a complicating retrobulbar syphilitic neuritis was excluded; and 3, bitemporal hemianopsia, which cannot be an accidental simulation by the loss of this part of the visual field because homonymous or binasal hemianopsia never is produced in the same way, but for which no satisfactory explanation can be given.

REICHARDT (153, **Studies of the brain**) claims that the cause of the reflex immobility of the pupil is not a disturbance in the reflex arch between the sensory and motor roots of the nerve, but is in diseases of the upper portion of the cervical spinal cord and about the fourth ventricle. Disturbances of the pupillary reaction can also originate in the cortex of the cerebrum. He distinguishes a tabetic, spinal "neurologic," and a paralytic, cerebral "psychiatric," reflex immobility of the pupil. The prognosis as to life may be said to be favorable when it is associated with meiosis, unfavorable when the pupils are moderately or widely dilated.

ELSCHNIG (143, **Indicanuria and diseases of the eye**) maintains that the demonstration of an abundance of indican in the urine is at the present time the best known symptom of the pathological breaking down of albumin in the organism, and that it should be taken into account in the etiology of certain diseases of the eye. He has found an increased quantity of indican in 40% of the cases of iridocyclitis in which no other disease could be detected in the organism to serve as its cause.

PICK and BIELSCHOWSKY (151, **Poisoning with methyl alcohol**) made histological examinations of the eyes and

central nervous systems of three patients who died of methyl alcohol poisoning. An extensive chromatolysis and breaking down of the fibrillary substance was found in the ganglion cells, uniformly distributed over all parts of the retina, in all of the cases. In addition there was in places, especially about the vessels, fatty degeneration of the optic fibers, yet these changes were unimportant in comparison with those in the ganglion cells. These injuries must be considered as primarily due to the poison, as the time was too short for the production of secondary degenerations. Signs of degeneration were also found in the ganglion cells of the central nervous system, but they were far behind those in the retina, both quantitatively and qualitatively.

For the treatment of methyl alcohol poisoning FOERSTER (146, **Diagnosis and treatment of methyl alcohol poisoning**) recommends stimulation of diuresis and diaphoresis by the administration of large quantities of fluid, sweat baths, lively movements (not rest in bed), and forced respiration in well ventilated rooms, when the patients are seen before the outbreak of the grave symptoms, as these measures accelerate the excretion of methyl alcohol, the same as they do that of ethyl alcohol.

FEHR (145, **Effect of salvarsan on the eye**) found that 217 out of 2636 patients examined before treatment with salvarsan had syphilitic conditions in the eye, 8¼%. The large number, 41, in which the condition was incidentally discovered, such as cases of advancing optic neuritis without visual disturbance, is striking. After the treatment with salvarsan 451 patients were seen again, and in 32 of these diseases of the eye had appeared in the meantime. Of these 12 had iritis, 3 chorioretinitis, 11 optic neuritis, and 6 pareses of the ocular muscles. It is difficult to compare the percentage of syphilitic eye affections after salvarsan with the percentage before the use of the drug, but Fehr thinks there is no material difference between them. He does not believe that the outbreak of these affections is to be blamed upon the salvarsan.

COUTELA (141, **Ocular accidents attributed to arsenobenzol**) gives a condensed account of the eye symptoms observed in the use of salvarsan. He considers it beyond all doubt that those which appear in the uveal tract after injections of

salvarsan are to be ascribed to the syphilis and not to the remedy. The same is true of optic neuritis. The objection that has been raised, that the papillitis comes on earlier than it is apt to do in syphilis, is untenable, as it is commonly observed between the first and the fifth month after infection. Besides, the clinical picture of the neuritis is not that of a toxic, but of an infectious condition. He quotes statistics to show that the frequency of inflammations of the optic nerve has not been increased since the introduction of salvarsan, as has been claimed, but that it remains the same. The same arguments are made to serve with regard to oculomotor changes.

CAUSÉ.

ANTONELLI (138, **Polyneuritis of the cranial nerves after treatment with salvarsan**) saw the onset of a left-sided facial paresis, a right-sided paresis of the abducens, and a bilateral papillitis of moderate degree, after a second injection of salvarsan twenty days after the first. The first injection was made two months after the primary infection. In addition to the above-mentioned troubles, the patient had paralysis of the right auditory nerve, and, some weeks later, paresis of the right oculomotorius. The symptoms passed away, leaving a paralytic strabismus and impaired vision with a slight optic atrophy. The cause was evidently a subacute meningitis. Antonelli believes that outbreaks of this nature are much more frequent after the use of salvarsan than after the classical treatment with mercury, and thinks it should therefore be used only in selected cases and with care.

CAUSÉ.

SATTLER (154, **Pathological Examination of a Case of Blindness after Injections of Arsacetin**) reports the case of a patient with pernicious anæmia who received intramuscular injections of arsacetin in the course of a month, the total amount of the drug used being nearly 5 grams. About six weeks after the first injection the vision failed and the papilla became pale, without the production of a central scotoma. During the next six weeks absolute amaurosis developed together with a genuine optic atrophy. A month later the patient died. The pathological examination revealed a chromatolysis and vacuolization of the ganglion cells of the

retina, atrophy of the nerve fibres, destruction of the medullary sheaths, and proliferation of the glia in the optic nerve, with less signs of degeneration in the optic tract. The papillomacular bundle was less affected than the rest of the nerve. The poison seems to attack particularly the peripheral segment of the third neuron.

III.—GENERAL AND EXPERIMENTAL PATHOLOGY AND TREATMENT. Reviewed by LOEHLEIN.

158. CONTINO. **Electric introduction of ocular anæsthesia.** *La Clinica Oculistica*, Jan., 1912, p. 825.

159. FRIEDBERG. **Discussion of Roemer's paper on anaphylaxis by means of lens albumin.** *Berl. ophthalm. Gesellschaft*, June 13, 1912.

160. GEBB. **Therapy of diplobacillus infection of the eye.**

161. IGERSHEIMER. **Experimental researches concerning syphilis in the eye.** *Ophthalmic Society of Heidelberg*, 1912.

162. ISSEKUTZ, B. v. **The synergism of local anæsthetics.** *Pflueger's Archives*, vol. cxlv., p. 448.

163. KRUSIUS, F. F. **Discussion of Roemer's paper on anaphylaxis by means of lens albumin.** *Ophthalmic Society of Berlin*, June 13, 1912.

164. KUFFLER. **Vitreous immunity.** *Ophthalmic Society of Heidelberg*, 1912.

165. ROEMER, P. **Anaphylaxis by means of lens albumin.** *Ophthalmic Society of Berlin*, June 13, 1912.

166. ROLLET and AURAND. **Experimental researches concerning the ocular infections caused by the gonococcus.** *Revue gén. d'ophtalm.*, 31, p. 97.

167. ZADE. **Antibodies of the cornea.** *Arch. f. Ophth.*, 82, 2.

KUFFLER (164, **Vitreous immunity**) sought to determine with how small a quantity of germs saprophytic infection could be produced in the vitreous With some, *e. g.* the vibrio Dunbar and the vibrio Metschnikoff, he was able to excite a serious purulent inflammation in the vitreous with from 200 to 1000 germs, but various other kinds of fungi in these numbers excited only very slight signs of inflammation or none at all. He then gives a number of experiments in which the transition of immune substances from the circulation into the vitreous was studied in actively immunized animals, which showed that complement-fixing substances were not demonstrable in the vitreous. Agglutinine and hæmolysine were occasionally found in the vitreous when the

index of the blood serum was higher than 1 : 10,000, and also when the index of the vitreous was not above 1 : 10.

KR.

The essential results of ZADE's (167, **Antibodies of the cornea**) researches are: The normal cornea of a non-immunized rabbit contains opsonine to staphylococci and avirulent pneumococci, yet to a much less degree than the blood serum. It plays no prominent part in the overcoming of corneal infections. The opsonine increases when the eye is irritated. The complement contained in the cornea of the pig is very slight. Foreign serum passes into the normal cornea of a rabbit properly prepared beforehand not less than 2½ hours after the introduction. A much greater effect follows an intravenous injection than a subcutaneous. Foreign serum enters more quickly and freely when the eye is irritated than when it is in a normal condition. Precipitine, agglutinine, hæmolysine, antitoxine, and perhaps bacteriolysine were demonstrable in the normal cornea when the rabbits had been previously prepared, especially when the eye was irritated by a puncture of the anterior chamber. He finds that the passage of antibodies into the cornea is considerably better than into the normal aqueous.

ROEMER (165, **Anaphylaxis by means of lens albumin**) says that it makes a great difference whether guinea-pigs are treated beforehand and reinjected with heterologous, or homologous lens albumin. When they have been treated beforehand with heterologous lens albumin, the reinjection, whether it be intravenous, or intraperitoneal, is followed by the typical fall of temperature that is characteristic of anaphylactic shock. But if the guinea-pigs are treated first with guinea-pig lens albumin the reinjection is not followed by this fall of temperature. Hence the theory that the lens albumin is to be considered an albumin to a certain degree foreign to the organism is not correct. The biological law applies to the lens albumin, that the organism, in the absorption of albumin from its own body, or from animals of the same species, controls the regulative devices which restrain the development of auto-anaphylactic bodies. The possibility is granted that this difference in the reaction of guinea-pigs to the use of heterologous and homologous lenses may be

only quantitative, for he has found in earlier researches that the formation of anaphylactic antibodies could be perceived in certain animals when the guinea-pigs were first treated with guinea-pig serum. In order then to be able to demonstrate the formation of lens albumin antibodies, which may perhaps be formed in guinea-pigs after previous treatment with homologous lens albumin, Roemer and Gebb instituted a new series of experiments to determine whether the formation of these auto-anaphylactic antibodies can be detected by febrile movements on reinjection of such prepared animals with small quantities of antigen. These have shown that the conditions must be somewhat different with lens albumin than with serum albumin, for the amount of stimulus required to induce febrile movements is relatively high, so that minute doses of lens albumin produce no effect on the previously treated animals. Hence it is not now possible to recognize with certainty the formation of auto-anaphylactic lens albumin antibodies by means of minute doses of lens albumin.

KÖLLNER.

On the other hand, KRUSIUS (163, **Discussion of Roemer's paper**) is of the opinion that there is an anaphylaxis produced in guinea-pigs by homologous lens albumin. As regards the increase of temperature referred to, this can also be produced by injections of salt solution and therefore forms no proof of the onset of anaphylaxis. In his opinion Roemer presents not an essential, but only a relative difference, for the reason that sufficiently fine methods are not as yet known by which to prove the onset of anaphylaxis. KÖLLNER.

FRIEDBERG (159, **Discussion of Roemer's paper**) objected to the interpretation Krusius had given to the anaphylactic febrile reaction. The supposed action of physiological salt solution, in which the albumin is suspended, to excite an increase of the temperature plays no disturbing part, for in such fever experiments the same volume of salt solution is always injected, and yet the temperature is influenced very differently, caused to rise or fall, according to the quantity of albumin contained in the solution. As regards Roemer's experiments concerning lenticular anaphylaxis in particular he considers them absolutely conclusive.

KÖLLNER.

IGERSHEIMER (161, **Experimental researches concerning syphilis in the eye**) reports the results of injections of mixed and pure cultures of spirochætæ into the blood current. Most of the injections were made into the carotid in order that as much material as possible should enter the eye. The primary symptoms produced are distinguished from those that appeared after a certain period of latency. The commonest primary symptoms were injection of the conjunctiva and of the ciliary vessels, together with white spots in the fundus. These white spots were due in the quite fresh stage to an acute chorioiditis and changes in the outer layers of the retina; in the later stage only the changes in the retina could be found. This condition gives a clear picture of the origin of the peripheral chorio-retinitis of congenital syphilis. As later consequences there were found several weeks after the injection: 1. A hard, superficial, ulcerated tumor of the lid containing spirochætæ, but no other bacteria, and pathologically presenting masses of leucocytes, thromboses of blood corpuscles, and infiltration of the vessel walls. 2. Tumor in the upper lid and in the plica semilunaris after an injection with a pure culture. Histologically: Masses of leucocytes, partly grouped, no changes in the vessels, no other bacteria. 3. Severe parenchymatous disease of the cornea with superficial ulceration. Total absence of the ganglion retinæ. 4. Typical parenchymatous keratitis after injection with a pure culture. This proves that there is such a thing as a primary parenchymatous keratitis. 5. Iritis, just beginning, with an exudate very poor in cells in the pupil. 6. Iritis together with spots in the inner layers of the retina. 7. Typical papules of the iris eight days after the injection of a pure culture. 8. Inflammation of the entire uvea, retina, and papilla. 9. Optic atrophy. Great changes in the retina; a focus of softening in the chiasm. 10. Temporal optic atrophy, pathologically the same as in tabes. 11. Optic atrophy at its very commencement after injection of a pure culture. Atrophy of the ganglion cells of the retina. No inflammation. The value of these researches lies in the possibility of finding pathologically nearly or quite identical changes in the early stage of syphilis in the human eye and of tracing their origin. KR.

ROLLET and AURAND (166, **Ocular infections by the gonococcus**) used rabbits for their experiments and found that contrary to the statement of Christmas, that the gonococci do not find suitable conditions for life in rabbits because of the higher bodily temperature (39° C.), the disappearance is rather due to phagocytosis. They inoculated cultures and toxines in the anterior chamber, the iris, ciliary body, vitreous, and optic nerve sheath. Inoculation of cultures or toxines in the anterior chamber resulted almost uniformly in a plastic iritis, which spontaneously recovered within three weeks, leaving posterior synechiæ. When a culture was inoculated, the iritis appeared by the next morning and was accompanied by a papillitis. Inoculation of the iris with cultures caused a plastic inflammation with hypopyon, which recovered within thirty-five days, leaving no synechiæ. Inoculation of the ciliary body was negative in two experiments. Inoculation of the chorioid with cultures produced no changes visible with the ophthalmoscope, but caused a granular degeneration of the outer layers of the retina. Inoculation of cultures or toxines in the vitreous caused a mild plastic iritis, with a hyalitis along the line of puncture, which was later absorbed; in several cases this was accompanied by an optic neuritis and an atrophy of the chorioid and of the outer layers of the retina. The introduction of bacteria or of toxines into the sheath of the optic nerve caused a severe papillitis that resulted in atrophy. The gonococcus seems to have a selective toxic action on the neuroepithelial elements of the retina and on the optic nerve, and its pathogenic effect seems to be chiefly due to toxines. At least bacteria were never found in the tissues of the rabbit's eye. The spontaneous recovery of infections of the anterior segment seems to be peculiar to rabbits, and may perhaps be due to a certain degree of immunity dependent on the high body temperature.

CAUSÉ.

GEBB (160, **Therapy of diplobacillus infection of the eye**) finds experimentally that certain coloring matters are able to affect diplobacilli in vitro and in the conjunctival sac of rabbits so that their growth in artificial media is inhibited. Clinical experiments likewise show a distinct action of the coloring matters upon the diplobacilli, in that affections of

the conjunctiva and lid caused by diplobacilli heal comparatively quickly under such treatment. Ulcers caused by diplobacilli are less amenable. These researches confirm to a certain degree the claims of Stilling in 1890 concerning the usefulness of certain aniline colors in ophthalmology, and give rise to the hope that better results in the treatment of diplobacillus infections may be attained by the development of this means than have been reached heretofore.

KR.

V. ISSEKUTZ (162, **Synergism of local anæsthetics**) ascertained by mixing different anæsthetics in various concentrations that antipyrin greatly increases the effect of cocaine, considerably that of eucain B, and slightly that of novocain. The latter increases the effect of eucain B considerably. Mixture of cocaine and eucain B, and of cocaine and novocain, showed a simple additional effect.

CONTINO (158, **Electric introduction of ocular anæsthesia**) concludes that alypin, cocaine, or holocain must be used if anæsthesia is to be obtained by means of iontophoresis. The inflammation caused by the electric introduction of holocain limits the choice to cocaine and alypin, the latter being preferable as almost quite unirritating. In this way complete anæsthesia of the skin may be produced for ten minutes, but if a few drops of adrenalin are added to the solution of cocaine, or of alypin, the anæsthesia may be prolonged to three hours. A current of five milliamperes is applied for six minutes, the positive pole applied locally, the negative held in the hand of the patient.

CALDERARO.

IV.—METHODS OF RESEARCH, REMEDIES, INSTRUMENTS, AND GENERAL OPERATIVE TECHNIQUE. Reviewed by LOEHLEIN.

168. ABADIE. **Ocular tuberculosis and its treatment.** *Société française d'ophtalmologie*, M y, 1912.

169. BEAUVIEUX. **Tuberculin and ocular tuberculosis.** *Ibid.*, May, 1912.

170. BENEDETTI. **Radioactive medicaments in ocular therapeutics.** *Ibid.*, May, 1912.

171. BIELSCHOWSKY. **A new prism apparatus.** *Ophth. Soc. of Heidelberg*, 1912.

172. BIRCH-HIRSCHFELD. **Some instruments for exophthalmometry and scotometry.** *Ibid.*, 1912.

173. DARIER. **Tuberculin and ocular therapeutics.** *Soc. française d'ophtalmologie*, May, 1912.

174. MOURADIAN. **The practical value of Wassermann's reaction in ophthalmology.** *Annales d'oculistique*, cxlvii., p. 1.

175. ROLLET and DURAND. **Measurement of protrusions of the eye.** *Revue générale d'ophtalmologie*, xxxi., p. 193.

BIRCH-HIRSCHFELD (172, **Instruments**) exhibited—1. A little apparatus with which to test the central color sense, composed of two disks movable upon each other. One disk contained test colors and mixed colors, the other apertures from 2 to 12*mm* in diameter. This apparatus permits of an instantaneous change of the test colors while the same distance is maintained. A scale at the back gives the size of the diseased area of the retina, corresponding to the size of the scotoma found in any case. 2. A modification of Priestley-Smith's scotometer to which certain improvements are made. 3. A simple and cheap device for the measurement of exophthalmus by means of photography. The outer margin of the orbit is marked by a line of pigment, or a strip of plaster. The head of the patient is fixed in a head-rest with a forehead support, the primary position of the eyes secured by fixing on a certain mark, and then a profile picture is taken with a camera at a certain distance. Just in front of the center of the pupil on the forehead support is a millimeter scale at the same distance from the objective as the apex of the cornea. The relations of the latter, as well as the outer margin of the orbit, to the scale may be secured by photography, or by tracing on tracing paper laid on the glass plate. The camera is then moved to the other side of the patient, who does not change his position, and the procedure is repeated. If a lens of seven diopters is used as the objective of the camera, the distance of the objective from the middle line is made 24*cm*, and the length of the camera is 36*cm*, a double linear enlargement is obtained, which facilitates the delineation and the measurement.

KR.

BIELSCHOWSKY (171, **A new prism apparatus**) described an instrument consisting of two double prisms on a stand that can be adjusted to the height of the eyes and the distance

between them. Each of the double prisms can be deviated seventeen degrees, and the angle of deviation can be read in degrees and meter angles. On one side of the prism frame are clips to hold spherical or cylindrical glasses, or a Maddox rod. On the other side colored glasses or little diaphragms may be placed. Below the arm carrying the prisms another arm may be introduced with an apparatus for stereoscopic exercises. The uses of the apparatus are: 1, to test the balance of the eyes; 2, to determine the amplitude of fusion; 3, to measure the angle of strabismus, both objectively and subjectively; 4, to test the sensory correspondence of the retinæ; 5, to excite binocular vision in strabismus; 6, to delimit exactly paracentral defects in the visual field; 7, to detect simulation of bad vision of one eye.

KR.

ROLLET and DURAND (175, **Measurement of protrusions of the eye**) describe an instrument which is essentially a modification of Hertel's exophthalmometer. It has only one mirror for each eye which reflects the apex of the cornea through a gauger so that the degree of change in the position of the eye can be read directly from the scale attached to a crosspiece. According to Rollet and Durand this instrument gives an absolutely reliable measurement, while errors of from 1 to 3*mm* are possible with Hertel's, through changes in the position of the observer. The authors give some clinical data as the result of studies with this new exophthalmometer. The normal position of the eye fluctuates between 12 and 14*mm*. The classical idea is confirmed that the eyes are small in hypermetropia, large in myopia. After tenotomy there is an exophthalmos up to 2*mm*. To "make a big eye" corresponds to an exophthalmos of 1 to 1½*mm*, and is presumably produced by a simultaneous contraction of the obliques. They present as a new fact the presence of exophthalmos in optic atrophy. Only one out of 19 such patients failed to have a protrusion of the eye, which ranged as high as 19*mm*. In some cases a higher degree of exophthalmos was found of the eye with an advancing atrophy. The exophthalmos of tabes, due perhaps to a hypotony of the recti, has been described many times. A protrusion of the eye to 22*mm* was measured in exophthalmic goitre. A protrusion of the eye was found

in 78% of the patients with nephritis. The instrument can also be used to measure senile exophthalmos.

CAUSÉ.

MOURADIAN (174, **Wassermann's reaction in ophthalmology**) discusses the practical value of Wassermann's reaction in ophthalmology on the ground of his observations in 245 cases. Although there are a number of affections of the eye that have long been known to be of certainly syphilitic origin, yet this reaction is in many cases of decisive importance. Its value is the same in syphilis of the eye as in syphilis of other parts of the body; it was found to be positive in 70% of the cases in which the diagnosis of syphilis was certain, in 45% of those in which it was probable, and in 23% of those in which it was uncertain. A positive reaction is of great value, a negative one of little. A negative reaction is obtained in 30% of syphilitics with ocular diseases; it shows neither absence, benignity, nor cure of the syphilitic process. A positive reaction indicates specially energetic anti-syphilitic treatment. It has in particular confirmed the etiology of parenchymatous keratitis as being due to hereditary syphilis, while the great number of negative reactions in cases of disease of the retina and uvea prove nothing as to the rarity of syphilis as the cause of these affections. The reaction was positive in 84.5% of the cases of parenchymatous keratitis, in 76.4% of ocular tabes, in 29.4% of iritis, in 18% of choroiditis, and in 37.9% of optic atrophy.

CAUSÊ.

BENEDETTI (170, **Radioactive medicaments in ocular therapeutics**) has treated 300 cases of trachoma with bromide of radium, almost exclusively by means of a radioactive ointment containing citrate of copper. The ointment was applied in the evening and Benedetti ascribes its beneficial influence to the radioactive constituents. Fifteen per cent. of the cases were cured within a month; forty per cent. in from forty to eighty days; the rest needed a longer time. Six per cent. proved obstinate.

CAUSÉ.

The treatment of tuberculosis of the eye was the subject of three papers at the May meeting of the Société Française d'Ophtalmologie. ABADIE (168) has obtained good results

by a method in which he gives iodgenal internally, applies inunctions of guaicol-lebertran, and has the patient eat from 60 to 190 grams of raw meat daily. The syrup of iodide and tannin is an excellent remedy in the scrofulous eye diseases of childhood. Raw meat as a food also plays an important rôle in the treatment of pulmonary tuberculosis. Injections of tuberculin are given at the same time in suitable cases. BEAUVIEUX (169) believes in very small doses in the beginning of tuberculin treatment. Starting with $\frac{1}{1000}mg$ of the dry substance he increases the dose within two months to $1mg$. He claims that non-progressive doses unnecessarily prolong the treatment and sometimes cause severe reactions. He reports good results in three cases of disease of the anterior segment of the globe and in three cases of choroiditis. DARIER (173) has treated forty-two cases with tuberculin in the past twelve years, twenty-five successfully, five unsuccessfully. In twelve the treatment could not be carried out regularly. The only complication seen was hæmoptysis in one case.

CAUSÉ.

V.—ANATOMY, EMBRYOLOGY, MALFORMATIONS. Reviewed by PAGENSTECHER.

176. ATTIAS, G. **Embryontoxon and arcus juvenilis.** *Arch. f. Ophth.*, 1912, lxxxi., 3, p. 505.

177. CARLINI, VITTORIO. **The construction and development of the zonule of Zinn.** *Ibid.*, lxxxii., 1, p. 75.

178. LOEHLEIN, W. **Anophthalmos with palpebral cyst.** *Zeitschr. f. Augenheilkunde*, 27, 5, p. 406.

179. PAGENSTECHER. **The causal genesis of malformations of the eye and congenital cataract.** *Ophthalm. Soc. of Heidelberg*, 1912.

180. v. SZILY. **The production of primary tumors in embryos and their importance in the genesis of glioma.** *Ibid.*, 1912.

181. UHTHOFF, W. **A case of very pronounced persistent pupillary membrane in both eyes.** *Klin. Monatsbl. f. Augenh.*, April, 1912, p. 476.

ATTIAS (176, **Embryontoxon and arcus juvenilis**) suggests as clinical characteristics by which the arcus juvenilis can be distinguished from the arcus senilis, that the latter is usually better marked above, the former below, and that the former has the sharper line of demarcation.

According to CARLINI (177, **The zonule of Zinn**) the zonule does not belong embryologically to the retina, but is differ-

entiated from the anterior part of the vitreous. The question whether the vitreous is of mesodermal or ectodermal origin, he leaves open.

PAGENSTECHER (179, **Malformations of the eye**) produced malformations of the anterior chamber experimentally by overfeeding pregnant rabbits with naphthalin. In some the development of the anterior chamber was completely inhibited, others had congenital posterior and anterior synechiæ. In one case the pupillary membrane had not been separated from the mesoderm of the cornea in an animal seven days old that had cataract. Inhibitions of development of the endothelium of the anterior chamber indicate relations to congenital internal ulcer of the cornea. True central, polar, and spindle cataracts were produced. An experimental malformation of the retina, a disturbance of the development of the zonule of Zinn, and an experimental malformation of the lens, rupture of the posterior capsule, were obtained in the last third of the pregnancy. Breeding experiments with a malformed animal gave normal progeny in only two generations.

KR.

V. SZILY (180, **Production of primary tumors in embryos**) has made a great many experiments to solve the question how far toxic influences take part in the origin of typical malformations of the eye. He does not confirm Pagenstecher's observations, and says that of the many observations made by that author there is only one of a truly typical malformation of the eye, and this may have been an accident. His verdict concerning the toxic origin of typical malformations of the eye is that it has not been proven to exist.

KR.

LOEHLEIN (178, **Anophthalmos with palpebral cyst**) demonstrated histologically that the cyst was a rudimentary eye. The posterior segment of the cyst contained a pigment membrane, the anterior segment a rudimentary retina. The time of the disturbance in development is placed by him before the time of the transformation of the primary into the secondary optic vesicle.

UHTHOFF (181, **Persistent pupillary membrane**) reports a case in which the persistent pupillary membrane interfered so much with vision that both eyes had to be operated on.

VI.—PHYSIOLOGY OF NUTRITION AND INTRAOCULAR TENSION. Reviewed by WESSELY.

182. VAN DER HOEVE. **Osmotic pressure and electric conductivity of intraocular fluids and blood serum of animals.** *Arch. f. Ophth.*, lxxxii., 1, p. 58.

183. KNAPP. **Influence of massage upon the tension of normal and glaucomatous eyes.** *Klin. Monatsbl. f. Augenheilkunde*, June, p. 691.

184. MAWAS. **The quantity of cholesterin in the normal aqueous, in the aqueous after paracentesis, and in the aqueous in senile cataract.** *Soc. d'ophtalmologie de Paris*, March 5, 1912.

185. WESSELY. **Study of the intraocular tension.** *Ophthalm. Society of Heidelberg*, 1912.

VAN DER HOEVE (182, **Osmotic pressure and electric conductivity of intraocular fluids**) concludes that there is no constant relation betweeen the osmotic pressure of the intraocular fluids and that of the blood serum in the sense of a hypertony of the fluids in the eye, but that these may be sometimes hypertonic, sometimes hypotonic. The intraocular fluid is often hypertonic to the arterial blood, and hypotonic to the venous. All these differences are probably explained by the slow renewal of the fluids in the eye and furnish no objection to the transudation hypothesis.

MAWAS (184, **Cholesterin in the aqueous**) found the amount of cholesterin in the aqueous ten times as great after puncture of the anterior chamber as in the normal, and he could not make out an increase in eyes with cataract.

WESSELY'S (185, **Study of the intraocular tension**) first series of experiments dealt with the dependence of the intraocular tension upon the blood pressure. Although in general the tension curve gives a true reflection of the blood pressure curve, yet deviations occur as soon as the change of blood pressure is caused by changes in the caliber of the peripheral vessels and these prevail in the eye. For the eye is to a certain degree an onkometer, and its tension depends not only on the pressure, but also on the fullness of the vessels. Faradization of the sympathetic, or intravenous injections of adrenalin, cause the tension of the eye to fall in spite of an increase of pressure in the carotid. The cases are clinically important in which the tension is increased by intraocular vasodilatation, as when the blood-vessels of the head and brain dilate with reduced pressure after the administration

of amyl nitrite, caffein, and antipyrin. Aside from the fact that these drugs may thus occasionally excite an attack of glaucoma, an interesting point is the harmony between the innervation of the vessels of the brain and of the eye, which goes to prove the importance of the movement of the blood in the physiology and pathology of the intraocular tension.

Other experiments were to determine the influence of the movements of the eye upon the intraocular tension. An increase of tension with every turn of the eye was demonstrated on one human eye, and on the eyes of monkeys. In the latter the least increase was made by the trochlearis, a greater one by the abducens, and the greatest by the oculomotorius, showing a marked difference in the influence exerted by the straight and the oblique muscles.

Finally Wessely made observations with Schioetz's tonometer. As the tonometer never measures the intraocular tension as such, but always a combination of this with the impressibility of the wall of the globe, and as only cadaveric eyes could be used, the calculations as to absolute heights of tension could have a claim to accuracy only if (1) the elasticity of the cornea of the cadaveric eye was the same as that of the living, (2) the elasticity of the wall of the globe was the same in all individuals, if the elasticity of the sclera was unimportant. But this is the case in neither respect. The elasticity of the cornea changes gradually in the cadaver, and the yielding or rigidity of the sclera has a very marked influence on the results obtained by the tonometer. Although the instrument is so valuable for the detection of relative differences of tension, yet the absolute values found by it must not be overestimated, and Wessely sees a danger that the diagnosis of glaucoma may be made and operations performed, because of high readings of the tonometer in the absence of other clinical symptoms. KR.

KNAPP (183, **Influence of massage upon the intraocular tension**) finds that massage of the eyeball through the lids with the fingers markedly reduces the tension of normal eyes within a few minutes, and that it takes the tension about three quarters of an hour to regain its normal height. In experiments on animals he could prove neither an increase of albumin in the aqueous, nor a rapid entrance of fluorescin, so

he supposes that the aqueous is regenerated in the same way that it is normally produced. In acute glaucoma, massage is usually ineffective; in mild attacks, or in glaucoma simplex, it almost always results in a decrease of tension after a quarter of an hour. The effect is greater and more lasting in eyes that have been operated on for glaucoma. If a considerable decrease of tension can be secured by massage several weeks after the operation the prognosis is good. But in exceptional cases massage may cause an increase of tension. Holocain reduces the tension in a large number of normal eyes, when it has been instilled several times.

VII.—PHYSIOLOGY AND PATHOLOGY OF THE SENSE OF SIGHT.

Reviewed by KÖLLNER.

186. BRUECKNER. **The galvanic excitability of the eye.** *Ophthalmological Society of Heidelberg*, 1912.

187. LENZ. **Central color-blindness.** *Berl. klin. Wochensch.*, p. 766.

188. SCHANZ, F. **Apparatus for observing the fluorescence of one's own eye, and the impairment of the vision by the fluorescent light.** *Ophthalmological Society of Heidelberg*, 1912.

189. SPOTO, G. **Colored protective glasses.** *Ann. di Ottalm.*, p. 165.

190. THIERFELDER. **The processes that take place in the retina.** *Beitraege z. Augenheilkunde*, 80, p. 1.

Central acquired color-blindness is always to be ascribed to a bilateral hemianopsia, according to LENZ (187, **Central color-blindness**), due usually to hemorrhages, or foci of softening. In the retrogression from total color-blindness blue is the first color to be recognized. Lenz denies the existence of a special center for the sense of color for various reasons. The color sense is probably only a higher, and therefore more easily injured, function of the visual center that is situated in the region of the calcarine fissure.

BRUECKNER (186, **Galvanic excitability of the eye**) has studied the question whether the condition of adaptation of the eye influences its galvanic excitability. He used fluid electrodes to carry the current and found, contrary to former writers, that adaptation to the dark produces an increase of sensibility. According to the strength of the current light phenomena may be perceived in the extreme periphery of the visual field, in the center, or in the region of the blind spot when the circuit is opened or closed. The increase in sensi-

tiveness in adaptation to the dark can be demonstrated only with the light phenomena in the center, or in the region of the blind spot; it is about double the sensitiveness in adaptation to light.

SCHANZ'S (188, **Apparatus for observing fluorescence**) apparatus for observing the fluorescence in one's own eye consists of a black box with a dark blue glass in one side wall and a mirror on its rear wall. If this box is held before the face like a stereoscope, in such a position that diffuse daylight from the window falls upon the eye after passing through the blue glass, the observer sees his pupil bright gray in the mirror, looking as if he had a ripe senile cataract. A great part of the visible rays of the diffuse daylight are cut off by the blue glass, while the blue, violet, and ultraviolet rays reach the eye. Only a portion of these rays act directly upon the light perceptive portions of the retina; another portion is transformed in the media of the eye, especially the lens and retina, into light of great wave length, so that these parts of the eye appear fluorescent. The fluorescence of the lens is so lively that the pupil appears to be bright gray. The phenomenon is particularly beautiful when the experiment is made with direct sunlight, or an electric arc-light.

KR.

THIERFELDER (190, **The processes that take place in the retina**) advances a new theory of vision, or at least of the processes that take place in the retina. In front of each cell lies a regeneration cell, the two connected by a "canal." The parts are surrounded by a photochemic neutral fluid. The inner limbs of the visual cells are permeated by the visual substance, which passes out again through the wall of the cell. The cell wall is the terminal apparatus of the nerves. "The different meridians are tuned to the different kinds of color molecules, like the rods of Corti's organ." The "violet meridian adjoins the red meridian." The inner limb of the visual cell represents an optical lens, the outer limb acts like a mirror. The rays that fall upon the inner limb are focused upon the outer limb, which reflects them. The visual cells and the regeneration cells appertaining to them have certain conflicting properties: the latter disperse light, the former collect it; osmotically, "the one forces the used visual mole-

cule outwards, the other, the regeneration cell, absorbs it." The visual substance is sensitive to light in all parts, yet yellow molecules are tuned only to yellow rays. Elsewhere the visual substance is not renewed, but undergoes regeneration only in the affected cells. Increase of light accelerates the circulation of the molecules, decrease retards it; this explains adaptation. The red molecule acts with double the energy of the last violet molecule; an explanation of Purkinje's phenomenon.

SPOTO (189, **Colored protective glasses**) has studied the chemical reaction of variously colored glasses used for protecting the retina from the action of the sun. He ascertained the degree of absorption of the differently colored glasses by means of photometric measurements, and studied the modifications which the different rays of the spectrum undergo under the influence of colored glasses, and determined the vision and light sense of different persons under the same conditions. He obtained the best results with Fieuzal and yellow glasses. This supports the claims made for the protective glasses, that they completely neutralize the violet rays, which are beyond doubt very tiring to the sensitive elements of the retina, and increase considerably the brightness of objects.

CALDERARO.

VIII.—ACCOMMODATION AND REFRACTION. Reviewed by KÖLLNER.

191. BEAUVIEUX and DELORME. **Bilateral paralysis of the accommodation.** *Arch. d'ophtalmologie*, xxxi., p. 163.

192. CABANNES and MARCAT. **Spasm of the accommodation and astigmatism of the crystalline lens.** *Ibid.*, p. 93.

193. HESSE, R. **The contraction of the pupil in near vision.** *Klin. Monatsblaetter f. Augenheilkunde*, June, 1912, p. 740.

194. KAHN. **Binocular union of one's own pupils.** *Arch. f. d. ges. Physiologie*, vol. 145, p. 249.

195. OLOFF. **A case of persistent paresis of the accommodation after diphtheria.** *Klin. Monatsbl. f. Augenheilkunde*, May, 1912, p. 551.

196. ROELOFS. **The association of accommodation and convergence,** *Inaug. Dissert.*, Amsterdam.

197. SCHUKOFF. **The relation of vision to errors of refraction.** *Diss.*, St. Petersburg.

198. UHTHOFF. **Demonstration of the process of accommodation.** *Trans. of the Ophthalmic Society of Heidelberg*, 1912.

UHTHOFF (198, **Demonstration of the process of accommodation**) had the opportunity to observe a child seven years old who had a total congenital irideremia with relatively good vision and good powers of accommodation. He was able to study the changes that take place in the anterior segment of the globe during accommodation, both when the latter was voluntary and when it was induced by eserine. Thus he was enabled to perceive the advance and swelling of the ciliary processes, the diminution in the circumference of the lens, slight irregularities in the marginal contour of the lens, and a narrow, bright circular zone along the equator of the lens.

KR.

A binocular union of one's own pupils can be readily perceived, according to KAHN (194, **Binocular union of one's own pupils**), if one looks into a mirror with parallel eyes. He then sees one cyclopic eye with two inner canthi, while the bridge of the nose appears as a crossed double image. He can then regard in turn the margin of the pupil, the corneal reflex, the posterior lenticular reflex, and make a number of interesting physiological observations. The sinking of the lens during strong accommodation, due to relaxation of the zonula, can be recognized from the sinking of the lenticular reflexes.

The contraction of the pupil in near vision may be produced wholly by the accommodation without connection with convergence, as is proved by HESSE (193, **The contraction of the pupil in near vision**). In four cases of unilateral paresis of the oculomotorius the patients were directed to fix on a near object with the paralyzed eye. The excluded healthy eye immediately assumed a position of marked secondary deviation, and yet its pupil contracted strongly. Experiments on himself also showed that attempts to accommodate with the eyes kept parallel, as with stereoscopic pictures, always induced contraction of the pupil.

C. OTTO ROELOFS (196, **The association of accommodation and convergence**) investigated the etiological importance of errors of refraction to the form of strabismus. In the first part of his work he details his studies on his own eyes concerning the nature, origin, intensity, and relaxation of the synergy of accommodation and convergence, which is not congenital, but is developed by practice and experience. The second

part contains his studies of the muscle balance of about 200 eyes. He finds that the errors of refraction are not directly accountable for the form of strabismus, as taught by Donders, but indirectly, through their influence upon the development of synergy of accommodation and convergence. With good binocular vision there is a tendency to orthophoria. With weak binocular vision hypermetropia is a frequent cause of esophoria, myopia of exophoria. When binocular vision for distance is absent in myopia, but present for near, esophoria may be produced in myopia from the development of a synergy of retinal stimulation and convergence.

B. P. VISSER.

CABANNES and MARCAT (192, **Spasm of the accommodation and astigmatism of the crystalline lens**) made careful studies in fifty cases concerning the spasm of the accommodation and the lenticular astigmatism, measuring very carefully the refraction of the principal meridians of each eye both before and after atropinization. They found differences in the lenticular astigmatism of between 2 and 3 diopters, with unlike behavior when the refraction was identical. They conclude that the eyes of children should always be atropinized for an examination of the refraction.

CAUSÉ.

BEAUVIEUX and DELORME (191, **Bilateral paralysis of the accommodation**) observed a bilateral paresis of the accommodation in a workman who had a burn of the first and second degrees of the face and the right forearm, while the function of the sphincter iridis was preserved. They are unable to give a satisfactory explanation of the origin of this paresis, but think it may possibly have been caused by intoxication from the use of compresses wet with concentrated picric acid which were applied for several hours immediately after the injury. In other respects the eyes were in a normal condition.

CAUSÉ.

OLOFF (195, **A case of persistent paresis of the accommodation after diphtheria**) reports the case of a sailor, 20 years of age, otherwise healthy, who had had a sore throat four years before, followed by a paresis of the accommodation which had persisted ever since. The possibility of some other cause

was taken into account, but it was rendered improbable by a careful examination.

SCHUKOFF (197, **The relation of vision to errors of refraction**) concludes from his studies that the impairment of vision and the degree of myopia are in no way proportional to each other. The vision may vary greatly with the same degree of myopia, and this may be the case in an individual patient. He likewise finds no relation between the vision of the unaided eye and the degree of hypermetropia. In one case of hypermetropia of 6 D. the vision was $\frac{20}{15}$, and yet the vision may be much impaired by a low degree of hypermetropia. A fixed proportion between the different kinds of astigmatism and the vision in like manner could not be established. The same power of vision is often present in various degrees of ametropia.

V. POPPEN.

IX.—THE MOTOR APPARATUS OF THE EYES. Reviewed by KÖLLNER.

199. FAGE. **Paralyses of the ocular muscles after traumatism to the margin of the orbit.** *Archives d'ophtalm.*, xxxii., p. 282.

200. FROMAGET, CAMILLE, and HENRI. **Latent nystagmus (nystagmus and strabismus).** *Annal. d'oculistique*, cxlvii., p. 344.

201. LAGLEYZE. **Congenital vertical strabismus.** *Arch. d'ophtalm.*, xxxii., p. 338.

202. LANDOLT. **Operative treatment of strabismus.** *Trans. of the Ophthalmological Society of Heidelberg*, 1912.

203. MUENCH, K. **The vermiform twitchings of the sphincter pupillæ.** *Klin. Monatsbl. f. Augenheilkunde*, June, 1912, p. 745.

204. ORDOP. **Hereditary nystagmus.** *Inaug. Diss.*, Leipsic.

205. TERRIEN. **Paralysis of the two externi following diphtheria. Treatment by serum therapy; cure.** *Arch. d'ophtalm.*, xxxi., p. 106.

206. WEEKERS. **Voluntary nystagmus.** *Ibid.*, xxxi., p. 86.

MUENCH (203, **Vermiform twitchings of the sphincter pupillæ**) considers these twitchings to be physiological and in no way pathological. The uniformity of the contraction of the sphincter is only apparently uniform because of the rapidity of the contraction. The sphincter is composed of some seventy or eighty physiological segments, each governed by a little nerve trunk, so that it has a certain degree of independence. The length of such a physiological segment corresponds to the length of the anatomical tissue element, the smooth muscle fiber. When the pupils are more than

moderately dilated, the light is weak, and the retina is adapted, the pupils have restless movements of a distinctly peristaltic character.

ORDOP (204, **Hereditary nystagmus**) reports a family in which fourteen cases of nystagmus occurred in four generations. The nystagmus occurred only in males, but was transmitted without exception through the females.

FROMAGET (200, **Latent nystagmus**) designates by this name, to correspond with latent strabismus, a form of nystagmus that is met with only under certain conditions. He reports the case of a man 21 years old who had nystagmus as soon as one eye was excluded from the act of vision, when reading, and when the eyes were turned far to one side. One eye was amblyopic, the other had perfect vision when both eyes were used, but only one-half when the bad eye was covered. When the amblyopic eye was covered a rapid nystagmus was set up, when the good eye was covered a similar nystagmus began, but with slow oscillations. Fromaget thinks that there is a function of the eyes, like that of the tendency to fusion, which prevents nystagmus and was done away with in this case by the occlusion of either eye. Probably there was a developmental fault in a center which is in close connection with the centers of coördination for convergence and for the associated lateral movements of the eyes. The simultaneous presence of a marked hippus shows that this hypothetical center is closely connected with the movements of the pupils. Fromaget believes that hysteria and simulation were excluded in this case.

CAUSÉ.

WEEKERS (206, **Voluntary nystagmus**) gives the details of a case of voluntary nystagmus, a condition that is rarely observed. The patient was a young man, 20 years old, who discovered when 7 years of age that he could produce a horizontal nystagmus in any position of his eyes. The oscillations were rhythmic, very frequent, and numerous. He had no strabismus. The pupils became small and oval simultaneously with the onset of nystagmus. Both eyes were normal, and there had never been any involuntary nystagmus. Voluntary nystagmus and voluntary strabismus are closely related; they are usually met with in persons who have a

finely developed muscular sense. In all cases the nystagmus is horizontal. Probably it is caused by a direct action of the will upon the supranuclear association centers of the lateral movements of the eyes.

CAUSÉ.

LAGLEYZE (210, **Congenital vertical strabismus**) had the opportunity to observe quite a number of cases of congenital vertical strabismus. Of the seventeen patients only two had strabismus deorsumvergens, the remaining fifteen had strabismus sursumvergens. The left eye was affected in fourteen, the right in three. Four were emmetropic, five were hypermetropic, two myopic, and three had hypermetropic astigmatism. The vision of both eyes was normal in eleven. Heredity did not seem to play any part. The most striking external sign in all cases was a torticollis. The ocular etiology of the wry-neck is very often not recognized in these cases, in which all tenotomies and orthopedic measures fail to give relief, while the position of the head is usually corrected simply by the wearing of proper prismatic glasses. Binocular vision is possible only when the head is held in an oblique position, and when the head is held upright the vertical strabismus becomes manifest. In strabismus sursumvergens the head is inclined to the shoulder opposite the affected eye, while in strabismus deorsumvergens it is inclined toward the same side as the eye, just as happens in paralytic strabismus. The inclination of the head is therefore to render possible the binocular act of vision, to compensate for the loss of muscular balance. In the majority of cases the trouble was a functional disturbance of the left superior oblique, in all but one the condition was congenital, and paralytic troubles were excluded in all. In four cases Lagleyze operated successfully, tenotomy of the superior rectus with or without advancement of the inferior, but in many cases the use of proper prisms is sufficient.

CAUSÉ.

E. LANDOLT (202, **Operative treatment of strabismus**) reports a case in which the patient had first a paresis of both interni and of the left externus. Advancement of both interni permanently restored the power of convergence. Thirteen years later the inferior rectus of the same patient became

paretic. Complete recovery followed advancement and resection of the affected muscle. The patient has perfect binocular vision, normal amplitude of convergence, and can do his work.

KR.

TERRIEN (205, **Paralysis of the two externi following diphtheria**) describes a case of bilateral paresis of the abducens six weeks after a slight attack of diphtheria. The velum was paretic at the same time, but the accommodation and the other muscles of the eye were not affected. The symptoms of paresis disappeared as suddenly as they came two days after the injection of *4ccm* of serum. Bilateral paresis of the abducens after diphtheria occurs very rarely, and the views concerning the efficiency of the use of serum in post-diphtheritic pareses are still divided.

CAUSÉ.

FAGE (199, **Paralyses of the ocular muscles after traumatism to the margin of the orbit**) shows that paralyses of the ocular muscles after contusions of the bony margin of the orbit are not as rare as appears from the small number recorded in literature. He reports 3 cases that have come under his own observation. In one a lasting paresis of the superior rectus of a boy of 10 years old was caused by a blow received from the head of another boy. He thinks that the fibers of the muscle were extensively torn. In another boy, 16 years old, a blow on the lower margin of the orbit caused a paresis of the superior oblique which never recovered completely. In the third case, a girl, 15 years old, received a blow that fractured the upper jaw and caused a paresis of the rectus inferior, which recovered in a short time. The cause of such a paresis may be: pressure upon the muscles by an effusion of blood into the orbit, partial rupture of the fibers of the muscle, hemorrhage into the sheath of the muscle, stretching of the muscle or inflammatory changes in the peripheral nerve fibers. The prognosis is very doubtful.

CAUSÉ.

X.—LIDS. Reviewed by KRAUSS.

207. CHRONIS. **Radical operation for the cure of trichiasis and entropion of the two lids.** *Arch. d'ophtalm.*, xxxi., p. 100.

208. ELSCHNIG. **Modification of Hess's operation for ptosis.** *Klin. Monatsblaetter f. Augenheilkunde*, May, 1912, p. 595.

209. KRAUSS, W. **The smooth muscles of the eyelid.** *Med. Klinik*, 17, p. 715.

210. MORAX and LANDRIEU. **Hyaline degeneration of the subconjunctival and epitarsal tissue of the two upper lids.** *Ann. d'oculistique*, cxlvii., p. 25.

KRAUSS (209, **The smooth muscles of the eyelid**) designates the system of smooth muscle fibers found in the anterior portion of the orbit as the membrana orbito-palpebralis musculosa. He shows that it has no direct relations with either the eyeball or the septum orbitale, while relations are present with Tenon's capsule, the levator, the conjunctival sac, the palpebral portion of the lachrymal gland, the accessory glands, and the smooth muscles of the veins passing through the anterior portion of the orbit. The membrana orbito-palpebralis musculosa has not only purely motor, but also vaso-motor and secreto-motor functions, which are of special importance to our understanding of the lid symptoms in exophthalmic goitre.

MORAX and LANDRIEU (210, **Hyaline degeneration of the subconjunctival and epitarsal tissue of the two upper lids**) saw a case of this nature in a woman 60 years of age, who had never before had any trouble with her eyes, and gave no antecedents in her history which could be brought into connection with her present disease. Both lids were so very thick that she could move them only a little, and they could not be everted without instrumental aid. Numerous subconjunctival hemorrhages had taken place in the course of the disease, which at this time had lasted three years. In the upper transition fold were large tumor-like hypertrophies which were placed upon and behind the thickened tarsus and crackled under the knife during the extirpation. A prolonged mercurial treatment was ineffective, but the operative removal of the hypertrophic parts brought about a material improvement. Embedded in the subconjunctival tissue were hyaline masses that had undergone granular degeneration, with numerous zones of ossification. Cases of hyaline and of amyloid degeneration of the conjunctiva are not very common, of the former about twenty cases have been recorded. The hyaline has frequently been considered an early stage of

amyloid degeneration; in a third of all cases the disease was bilateral. The pale yellow, irregular, hypertrophic appearance of the conjunctiva, and the thickening and broadening of the tarsus that can be felt, are to some degree characteristic. Half of the cases that have been met with were also cases of old trachoma. The majority of the patients were from 25 to 30 years old, and the commencement of the disease in the 56th year is exceptional. The diseases from which it needs to be differentiated are lymphoma, leucosarcoma, cicatricial trachoma, and certain forms of tuberculous conjunctivitis and specific tarsitis.

CAUSÉ.

CHRONIS (207, **Radical operation for trichiasis and entropion**) recommends the thinning of the tarsus, which was devised by his father and has recently been described as a new operation under the name of tarsoleptinsis, and emphatically claims priority. The skin of the lid is incised 2 to 3*mm* above the free margin, the palpebral muscle is excised so as to lay bare the tarsus, which is then thinned with a knife, and sutures are introduced which are tied at the ciliary margin. Finally an intermarginal incision is made.

CAUSÉ.

ELSCHNIG (208, **Modification of Hess's operation for ptosis**) uses a piece of the fascia lata for the fixation of the lid skin and has obtained good cosmetic results in this way. He also obtained an excellent result in a case of congenital ptosis through the advancement of the levator.

VIII.—Lehrbuch der Augenheilkunde (Text-Book of Ophthalmology). By AXENFELD, with the collaboration of Bach, Bielschowsky, Elschnig, Greeff, Heine, Hertel, v. Hippel, Krückmann, Oeller, Peters, and Stock. Third edition, pp. 761. Jena, G. Fischer, 1912. Price 15 Marks.

This new edition has been revised and enlarged; new illustrations have been added; the text has been somewhat rearranged.

The book, written by those to whom the teaching of Ophthalmology in the German Universities is intrusted, is a striking tribute to the scientific character of this instruction. The wealth and excellence of the many colored illustrations constitute one of the most important features of the book.

A. K.

IX.—Electricity in Diseases of the Eye, Ear, Nose, and Throat. By W. FRANKLIN COLEMAN, Chicago. Pp. 595. The Courier-Herald Press, 1913.

After introductory chapters on the Physics and Therapeutic use of Electricity, the various diseases of the parts described in the title are taken up in turn, and the successful manner in which they were treated by electricity is described. In the part on the eye, aside from the generally accepted value of the galvano-cautery in corneal ulcers and keratoconus, and the extraction of intraocular particles of steel with the giant or hand magnet, no evidence is brought of the value of electricity in the treatment and cure of practically all other affections.

A. K.

X.—Diseases of the Eye. By J. HERBERT PARSONS, London. Second edition, pp. 684. Philadelphia, P. Blakiston's Son & Co., 1912. Price, $2.50 net.

This text-book, which was favorably reviewed in these ARCHIVES when it was first published, now appears in a second edition, with more illustrations and a revised text. It represents a well-balanced, scientific treatise, and occupies one of the first places among the medium-sized text-books on the eye.

A. K.

XI.—Outlines of Applied Optics. By P. G. NUTTING, Assoc. Physicist, Bureau of Standards, Washington, D. C. Pp. 234, with 73 illustrations. Philadelphia, P. Blakiston's Son & Co., 1912. Price, $2.00.

This little book deals with optical instruments and optical measurements, which the author defines as belonging to optical engineering, a branch practically untaught in any university. It has been prepared for those who design instruments, measure colors, examine eyes, and identify illuminants. These are all important subjects with practical bearing, which many will find worthy of careful study.

A. K.

XII.—Die Sehschärfe des Menschen und ihre Prüfung (Human Visual Acuteness and its Determination). By Dr. LEOPOLD LÖHNER, Graz. Pp. 136. Leipzig and Vienna, Franz Deuticke, 1912. Price, 4 Marks.

This monograph treats the important chapter in physiologic optics on acuteness of vision. For its determination no less than 73 test-objects and charts have been devised, which to the author's mind proves that the question has not been finally solved. At the same time he wishes to approach the subject on a broader plane, and bases his study on investigations on the physiologic foundations of visual acuity. He is inclined to regard acuteness of vision as the visual act of the dioptrically normal eye. Dot-objects and letter-objects should not be considered of equal value. Dot-objects furnish a valuable means to discover certain variations of vision which are not recognized by letter-tests; for ophthalmological purposes they can, however, not be made practical on account of many possible errors.

A. K.

XIII.—Diagnostik der Farbensinnstörungen (Diagnosis of

Color Blindness). By Professors STARGARDT and OLOFF, Kiel. Pp. 45. Berlin, Julius Springer, 1912. Price, 1.80 Marks.

The authors of this unusual little book succeed in bringing in 46 pages everything essential in the examination for color blindness or color deficiency. It well deserves an English translation, and ought to be a "vademecum" for all who are interested in railroad or similar examinations.

The first part of the book deals with the two theories of color perception and its anomalies. The second part gives the diagnostic value of the different methods of examination. The authors conclude that an absolute diagnosis of color blindness or color deficiency can be established only by the use of Nagel's "Anomaloskop." For cursory examinations, Stilling's test-cards are sufficient and answer every purpose, while those of Nagel are apt to be deceptive. Absolutely unreliable are Holmgren's tests and any tests which represent an imitation of railroad signals.

E. SCHALCK.

XIV.—Die Anomalien der Skleralspannung (Anomalies of Scleral Tension). By Dr. HUGO STRANSKY, Brunn. Volume I. Pp. 243. Leipzig and Vienna, Franz Deuticke, 1912. Price, 6 Marks.

Scleral tension depends not only on intraocular pressure, but on the rigidity of the sclera itself. This scleral rigidity the author regards of great importance in glaucomatous conditions. He therefore has set to work to study the laws which govern scleral tension, and believes to have discovered the true nature of simple glaucoma, and that a number of physiologic and pathologic conditions and processes in the eye stand in causal relation to scleral tension and its variations. This Volume I. treats of glaucoma inflammatorium, the senile eye, the scars of the sclera, and glaucoma simplex (Part 1).

A. K.

NOTICES.

XIITH INTERNATIONAL CONGRESS OF OPHTHALMOLOGY.

(St. Petersburg, July 28–August 2, 1914.)

1st. Circular St. Petersburg, December, 1912

Dear Colleague: To prepare for the Congress which stands under the protection of His Majesty the Emperor, a central bureau was founded in St. Petersburg.

The members of this bureau are: the professors of ophthalmology from our universities, representatives of ophthalmological societies, as well as oculists from several important towns of the Empire.

The central bureau took care to invite renowned oculists from all civilized states as corresponding members. All those members form the international organization committee.

According to the usual custom we have conferred the honorary presidency on our eminent colleague, Professor Arnaldo Angelucci (Naples).

In the regulation of the XIIth Congress, the rules adopted at the Xth and XIth Congresses have been taken into consideration.

Until now the official languages at Congresses have been English, French, and German. In Naples, Italian and Spanish were added. This time, we unite with them the Russian language.

We request the authors who want to make a communication, to express the principal ideas, thesis, and conclusions of their work in French; otherwise it is impossible to discuss, which is the principal aim of the Congress.

Will you kindly transmit in time your participation in the Congress, directly or through intervention of corresponding members from your country? We hope that you will take an active share in the work of the meeting in St. Petersburg.

All information and circulars which the organization of the Congress publishes will be sent to you by one of the representatives of your country.

With collegial greetings

Professor *L. G. Bellarminof.*
President of the central bureau and of the international organization committee.

Professor T. GERMANN, the General Secretary, wishes to announce that it is only essential for those speaking in Russian, Italian, and Spanish to add an abstract in French to their discussion. The subjects selected for discussion are:

I. The Etiology of Trachoma, and
II. The Nutrition of the Eye.

Professor AXENFELD will deliver the address on the first theme, while the second will be treated by Professor ANGELUCCI. Lieutenant-Colonel HENRY SMITH, of Amritsar, India, will speak on his method of cataract extraction.

XVIITH INTERNATIONAL CONGRESS OF MEDICINE.

(LONDON, AUGUST 6 TO 12, 1913.)

We are asked by the Secretaries of the Section of Ophthalmology to recall to our readers the following Rules of Congress:

The titles of all Independent Papers should be announced to the Central Office of the Congress by the 30th of April, 1913. Any papers offered after the 30th of April, 1913, will be placed upon the agenda only after the discussion of those which have been announced before this date and have been chosen by the Council of the Section. No paper will be accepted unless the title has been received by the Secretaries of the Section before the 1st of July, 1913. The text of every communication must be typewritten. Independent Papers may be written in English, French, German, or Italian.

B. SECTION IX.—DISEASES OF THE EYE.

President: Sir HENRY SWANZY, M.D., F.R.C.S.I.
Secretaries: E. TREACHER COLLINS, F.R.C.S.
J. B. LAWFORD, M.D., C.M., F.R.C.S.
GEORGE MACKAY, M.D., F.R.C.S.Ed.
LOUIS WERNER, F.R.C.S.I.

Thursday, August 7th. 1. The Pathogenesis of Chronic Uveitis, with the exception of syphilitic, tuberculous, and sympathetic forms, by Professor Dr. ERNST FUCHS, of Vienna, and Professor DE SCHWEINITZ, of Philadelphia, U. S. A.

Friday, August 8th. 2. Operations for Glaucoma with special reference to the comparative results of iridectomy and its newer substitutes by Professor LAGRANGE, of Bordeaux, and Professor PRIESTLEY SMITH, of Birmingham.

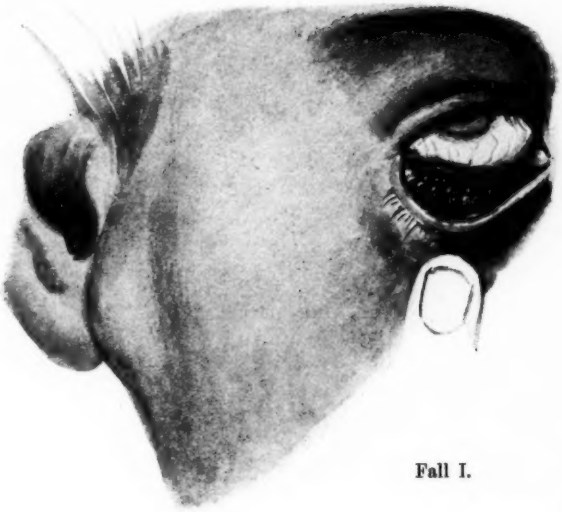
Saturday, August 9th. 3. A Demonstration or a Discussion on a theme not yet decided upon.

Monday, August 11th. 4. Diseases of the Eye caused by improper light action, by Professor CARL v. HESS, of Munich, and JOHN H. PARSONS, F.R.C.S., of London.

Tuesday, August 12th. 5. Anaphylaxis in its Relation to Ophthalmology, by Dr. V. MORAX, of Paris, and Dr. AUREL v. SZILY, of Freiburg-i.-Br.

On September 18, 1913, Professor JULIUS HIRSCHBERG will celebrate his seventieth birthday. Those who have been his pupils, intend to honor the eminent ophthalmologist by presenting him with an artistic tablet, and request contributions toward a fund for this purpose.

These contributions should be sent to Dr. W. MÜHSAM, Berlin, W. 30, Motzstrasse 79, Germany.



Fall I.

Fall II.

Krusius-Clausen, Konjunktivitis Parinaud.

VERLAG VON J. F. BERGMANN IN WIESBADEN.

Fig. 1.

Fig. 2.

Hack, Ein Fall von Eversion des Pigmentblattes der Iris.

Verlag von J. F. Bergmann, Wiesbaden. Königl. Univ.-Druckerei H. Stürtz A. G., Würzburg.